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TRANSACTIONS

OF THE

Thirty-fifth Annual Meeting

OF THE

American Laryngological Association

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TRANSACTIONS OF THE THIRTY-FIFTH ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION,

HELD AT WASHINGTON, D. C.,

May 5th, 6th and 7th, 1913.

THE PRESIDENT'S ADDRESS.

BY GEORGE A. LELAND.

Fellows of the American Laryngological Association and Guests:

To this the thirty-fifth annual meeting of the oldest national laryngological society in the world, it is my bounden duty and great privilege to most cordially welcome you, and I do it with due appreciation of the great honor which you have conferred upon me by adding my humble name to that noble list of my predecessors in this high office, the Nestors of our specialty in America. For, in common with some of them, I consider it the highest honor which can come to any man in our branch of medicine; and I may add it is one which I had no right to expect, having apparently deflected from strict laryngology to the study and practice of what should be considered its most important branch.

And I am very glad and proud indeed to have this opportunity to address you as man to man directly and not as one isolated by high elevation and separate, but speaking naturally and with my own voice as a human being trying to co-operate with other human beings in a common service* for the good of suffering humanity, for the prolongation of life, and the saving of it sometimes, and for the increase of the comfort and efficiency of those who entrust themselves to our care, which is sometimes better than life itself, or that which often makes life worth living.

*With apologies.

And I wish to express to you on this occasion, although nineteen years after, what an honor I considered it and still do with increasing appreciation to be admitted to this notable scientific body at all, to this club of good fellows, of honorable gentlemen, all working in the same cause, the advancement and perfection of the knowledge of all that concerns the upper —no, all the air passages—the Senate, as it were, of matters laryngological on this hemisphere.

And yet again I wish to say that I am bound in great obligations and gratitude to our most efficient and hard-worked secretary, to whom is due more than any other the compilation of our scientific programme and the anticipated success of our meeting. I want to say right here a word in his behalf. It is not, I am sure, half appreciated by any of us, except perhaps by those who have been secretaries, what an enormous amount of time and labor this office demands, and what a great tax it is upon a busy practitioner. I have heard that it has been said that it was hoped we would not work our present secretary as we did our last one, who was an all too willing slave, and who sacrificed himself to the good of our beloved association, to which he was devoted. Do you know that more than one-third, perhaps as much as one-half, of the employment of the private stenographer of our present secretary has, during the last six months, been on the necessary work of this association?

It seems to me it would be well to make some provision for the extra clerical work which devolves upon him. The transactions to issue as the result of this meeting with its twenty-seven or twenty-eight papers and seven candidates' theses will make one of the largest volumes in our set. The proof reading devolves on the secretary. Cannot we relieve him of some of this hack work either by paying to have it done, or perhaps by volunteering to do some of it ourselves? Since our incorporation makes it necessary to have a New York man for secretary, we must, therefore, perforce, have a busy one; not like the quacks, osteo-quacks and homeo-quacks, busy by assumption, but really and actually busy; hence the necessity of re-

lieving him of as much of his labor as possible. We choose him for his brains, not his muscle; for his guiding hand, not the horny hand of toil.

The more I have read of the history of our association and become imbued with its spirit the more honor and respect and admiration I have for those twenty-one gentlemen who met in Buffalo, June 3, 1878, and projected our cherished association. And of these founders we rejoice that after thirty-five years we still have on our list of active fellows four and on the retired list one. And there are three others still living, one of whom is yet in active practice in a wider field; so that of those who founded the world's first national association for the study of laryngology there are still eight living. And, as is so often the case, we can well say they laid the foundation for a structure greater than they knew, or could, perhaps, even imagine. And we must admire their intrepidity and determination, for in those early days the use of our mirrors was looked upon with doubt, disbelief, and ridicule. Dr. Elsberg in the first annual address expressed this, as quoted by Delavan in his masterly address in 1894, when he said: "The road has been a rugged one, and few of you can realize to-day the up-hill work, the obstacles and obloquy encountered." Even in the writer's memory, back in 1883 or 1884, how oracular was the advice of his medical and surgical friends that exclusive throat practice would never be successful; and more than one general practitioner then turned up his nose and, with a contemptuous smirk, said, "Playthings," when the laryngoscope was laid out for examination of a tuberculous larynx. And yet one of them had to acknowledge its efficacy when euthanasia was brought about by swabbing out such a larynx.

The field has widened enormously during the last thirty-five years. One of our early presidents deprecated the addition of the nose to our subject of laryngology, but the discovery of cocaine made it inevitable that it and its adnexa should be added, and studied and mastered. And since the ear is also an accessory cavity, nine-tenths of whose diseases originate in the mucous membrane of the nose and the naso-pharynx, the

preventive measures against as well as treatment of aural disease are to be carried out in the regions falling under the jurisdiction of the American Laryngological Association. This was brought out in 1883 in a paper wonderfully erudite for that time, written by Dr. Beverley Robinson, one of our former members, and published in the Transactions of that year.

Preventive medicine is the great subject prominently brought to our attention at this meeting, because of the great fame of the President of this Triennial Congress in this regard. His phenomenal work in Cuba and in the Canal Zone, where he has made the pestilential tropics inhabitable, is one of the wonders of the world; and the name of Gorgas will ever shine as one of the bright and particular stars in the galaxy of American medicine. This is attested by the fact that whereas during the French occupation daily a train load of dead was taken to that most populous of graveyards at Ancon, so numerous were the victims of tropical zymotic disease, that records could not be, or at least were not, kept. Now the death rate is reduced to that of the most healthful parts of the earth. But this is characteristic of American medicine. While Colonel Gorgas has saved his tens of thousands, it falls to each and every one of us to be enlisted in the cause of preventive medicine. And this is the spirit of our association and of our profession, my brethren, to prevent as well as to cure, to destroy our business and deplete our pockets for the good of suffering humanity. So now the modern Samaritan on the Jericho pike not only heals the sufferer by the wayside, but seeks out and destroys the cause of his malady, whether intrinsic or extrinsic, so that there will be fewer sufferers from the same cause for the exercise of the benevolence, skill or greed of other Samaritans.

Our founders builded wiser than they knew when they chose the throat as the field of their specialty, for here, as shown by modern bacteriology and clinical observations, is the port of entry of many of the diseases; nay, we might almost say most of the diseases to which poor human flesh is heir. The New York daily which said: "The nose is the source of all our

woes," was not far from right, but would have been nearer right if it had said the throat and nose. For it is well established that here may enter through the lymphatics of the fauces and nose micro-organisms which cause tuberculosis, nephritis, endocarditis, sepsis, cerebrospinal meningitis, acne, erythema nodosum, erysipelas, etc., etc., etc.; and by extension up the Eustachian tube, aural disease of all sorts leading to deafness, pyemia, meningitis, intracranial abscess, and so on and on. And it is in our province of preventive medicine to obviate all this, to put the upper air passages into the best possible shape, and to teach our patients to use them. I repeat, to teach the proper and continuous use of them, for evidently this is a most important part of our work, since this gives nature the chance to use the wonderful measures she has provided for the protection of our organism.

It is not necessary to prove this by going into the physiology of the nose and of the faucial lymphatics before this audience, but it is desired to emphasize the contention that the laryngorhinologist is the best and most rationally obvious aurist, because in his hands lies the prevention of those pathological conditions which bring about so much pain, distress, inefficiency, misery and danger, and because in his region is the best point of attack for either acute or early chronic middle ear disease. In a speech at the banquet of the Ninth International Otological Congress in Boston last August, one of the first otologists in America and hence of the world, advocated the study of pure otology untrammeled by other branches of the upper air passages. But is is evident to the members of the American Laryngological Association without doubt that if the nose and naso-pharynx could be put into proper shape and continuously used, the occupation of the aurist would be gone except for the effects of traumatism and perhaps for the infections, though it is evident these would be largely reduced except again for invasions of the internal ear from such affections as mumps, pregnancy, and some forms of meningitis.

It has been said above that in our region lies the best point of attack for acute otitis media, and it may not be out of place

to say that many cases of acute inflammation of the middle ear may not only be prevented, but also aborted even when the drumhead is red or even bulging, by relieving the congestion and engorgement of the naso-pharyngeal lymphatics, which means the adenoid operation especially including the fossae of Rosenmueller. This has been the writer's practice for twenty-five years in private when possible, and in hospital practice when opportunity offered. Many inflamed and acutely distended rumheads have been saved perforation by the needle or otherwise with all the annoyance of discharging ears consequent thereon, because drainage was established from within.

And so also in the region of our activities are to be found the ports of entry for infections of the cervical glands. And *we* know how universally we find that these unsightly tumors are preceded by diseased adenoids and tonsils when acute, and how equally frequently chronic indurations here are accompanied by chronic disease of these lymphatic structures. In fact, in nowise can here the diagnosis be complete unless the condition of the tonsils and adenoids be thoroughly ascertained; and yet how often is it the case that the general surgeon performs his extensive dissection for the removal of these glands and leaves the morbid source untouched and probably unknown.

So that here again is our work preventive, if it results in the removal of intracervical disease before the extracervical indurations have broken down and become fluctuating. And by the same token the most effective treatment of early cervical adenitis is the thorough ablation of our old friends, infected tonsils and adenoids. Moreover, it is by no means necessary to consider these glands tubercular even though the pathologist may find a few bacilli in their substance. Dr. G. Sims Woodhead in his illuminating article on the "Channels of Infection"* showed conclusively that these glands can take care of and destroy infectious matter passing down the lymphatic chain, including even tubercle bacilli. Thus does

*Lancet, October 27, 1894.

preventive medicine come into our province where we seek to make effective nature's abundant protective measures.

And so from our restricted region the field is ever broadening. Headaches, neuralgia, that terrible tic douloureux which inflicts so much misery and leads even to suicide—these often find their origin in the peripheral distribution of the fifth nerve in the nose and its accessory cavities, when pressure from retention of pus or from contact can very often be found if sought, the relief of which would obviate the necessity of operations on the nerve, and especially the removal of the gasserian ganglion, which is so often not successful permanently, or even for a short time.

Moreover, from the original demarcation of laryngology we have not only gone upward, but the introduction of the Kierstein's method of direct laryngoscopy has taught us to go downward, so that not only the larynx but the trachea and bronchi are open to our vision. And, furthermore, behind the larynx the esophagus, and even the stomach, can now be penetrated by illuminating tubes and ingenious instruments, the products of the consummate skill and inventive genius of Killian, of our corresponding list, and of Jackson and Mosher and other active fellows of our great association.

And now as a final complaint to us, the studies of the pituitary body, i. e., the hypophysis cerebri, have shown the desirability of its removal; and the most obvious way and the most usual is through the nose, the operation of resection of the septum having shown the best way to enter the sphenoid and to reach the gland by removal of its roof.

So that it can now be said without fear of contradiction that the confines of laryngology as laid out by our noble founders thirty-five years ago have been extended till they now embrace most of the upper half of the body—from the umbilicus to the crown of the head. Truly, in the language of that doughty warrior Commodore Perry, "We have met the enemy and he is ours." And it may be added that the aggressive spirit of progress of the American Laryngological Association will advance in the future as in the past.

When other obstacles to advancement appear we shall quote again from one of our greatest generals, who in 1863 was called "Unconditional Surrender Grant," "I propose to move immediately upon your works."

And yet in the accurate knowledge and prophetic vision of our first President much of this field was outlined in his definition of the boundaries of our specialty. To quote from his address, p. 36,* "The throat specialist of to-day is called upon to treat all these diseases, and more, viz., affections of the larynx and trachea; of the root of the tongue, valleculae and hyoid bone; of the tonsils, palate and uvula; of the pharynx, pyriform sinuses and oesophagus; of the pharyngo-nasal space, nasal passages and frontal sinus; of the thyroid body, parotid and sublingual glands, and of the areolar tissue and other structures that make up the anterior portion of the neck.

"Besides, as the mucous membrane of the throat is continuous with that of the whole alimentary tract on the one hand, and that of the bronchi and their ramifications to the air cells in the lungs on the other, and through the pharynx and nasal fossae extends to the sphenoidal, ethmoidal and maxillary sinuses, through the lachrymal duct to the conjunctiva, and through the Eustachian tubes to the mastoid cells and tympanum—certain diseases of these regions must sometimes be considered by the laryngologist in addition." And then he makes this most pertinent and forceful remark: "I need not here dwell upon the fact that the connection between throat diseases and other diseases of the body is so intimate that no one is competent to be a throat specialist without a previous thorough medical education and considerable general clinical experience." It is to be feared that this is not always heeded and acted upon.

During the past year no startling discovery has come to notice in our specialty, but in the process of sifting out the chaff and conserving the grain notable advance may be recorded toward perfection in endoscopy of and treatment in

*Transactions of 1879.

the bronchi, esophagus, nose and naso-pharynx. Vaccines and serums and radium are being tested and exploited, so that in the near future we may expect them to take their proper places in our therapy. The great mountains of tuberculosis and of malignant disease yet remain unconquered, though the former in early stages seems to be more or less amenable to climatic and diatetic treatment. The early surgical removal of localized malignant disease seems, however, to be making some progress.

Much has been said in the thirty-four preceding presidential addresses on the subject of education, the education of the undergraduate, how much of laryngology could be crowded into his short and overcrowded curriculum; of the post-graduate, how much could be shown and taught him in the few weeks or months he could devote to the subject—i. e., how much of a near-specialist we could make of him.

But there is a large class still which has not been mentioned—the general practitioner. And it is, it seems to me (aside from the pleasure of renewing delightful acquaintance), the one great object of these annual meetings to discuss questions and measures of utility and importance, and to put out our conclusions in an attractive, concise and authoritative form so as to demand the attention of every doctor. And to this end our papers should be widely spread through journals of general medicine, and not embalmed in our volumes of Transactions only, valuable though they may be and are. In saying this I am not unmindful of the great circulation of the Journal of the American Medical Association and of the two great New York journals, but I suggest still wider dissemination, so that dangerous conditions may not become fatal because unrecognized, with a laryngologist or bronchoscopist near at hand, but not consulted.

"Of making many books there is no end, and much study is a weariness of the flesh," was said long before modern medical writings were even conceived of; and yet it is our duty and privilege to add to them, to publish the results of our pioneer work for the guidance of those whose field is

less restricted. And for the busy practitioner the short, concise article dealing with the commoner, yet sometimes puzzling, and obstinate maladies to be found in the upper air passages, which he may almost daily meet with, is more acceptable than the long, learned, scientific and often largely historical monograph on those diseases which even the specialist rarely sees. To be a guide, a teacher, an aid, it is well to write clearly, to the point. To show our learning, our industry, the labor of our research and the extent of our experience we can afford to be comprehensive, discursive, prolix and ponderously scientific, but then our readers will be largely, if not exclusively, specialists like ourselves.

At the beginning of the nineteenth century all of medical knowledge, as well as the whole range of human knowledge of that time, was compassed by the brain of one man, *Dr. Thomas Young, of St. George's Hospital, and just a century ago this year (1813) was published his first book, "Introduction to Medical Literature, Including a System of Practical Nosology," a work of great labor and of great assistance to students and investigators. But at that time the whole of medical literature would not equal that of any one of our modern specialties, the mastery of which, together with a necessary working knowledge of general medicine or surgery, taxes severely enough the minds of even our greatest intellectual prodigies.

It is my sad duty to record that three of our fellows have joined the "innumerable caravan" during the year.

Dr. C. E. Bean, of St. Paul, was admitted in 1880. He seems to have contributed a few articles in the early years of his membership, and was a genial fellow, though latterly little known.

Dr. James E. Newcomb, our last president, died two and a half months after he relinquished the office. At the meeting in Philadelphia, 1911, it was, alas! too obvious that he was in failing health, and we all heartily approved of the action of the nominating committee in conferring upon him what

*See Peacock's Life.

he considered "the highest honor in the power of American laryngologists to bestow," and hoped it was not too late. He served us faithfully and assiduously for eleven years in the office of secretary, during which time our membership had doubled. Learned, skilful, philanthropic, influential, a hard worker and genial good fellow, his memory will ever be enshrined in the hearts of those who had the good fortune to know him, and especially in your hearts, my brethren, of the American Laryngological Association.

And last Tuesday, the 29th of April, passed on our former president, Dr. C. H. Knight. He came into the association in 1885, served as secretary 1889 to 1895, and was president in 1896. He was a hard worker, a learned professor, contributed much to our Transactions, and was withal a splendid, kindly, genial gentleman. It was always touching to me when I heard his New York confreres refer to him as "Charlie Knight"; it showed their familiar, affectionate regard.

Newcomb and Knight!—two great souls have passed on to their well deserved and abundant reward, and have left a desolation in the ranks of our most highly esteemed, great association. I cannot better express what is in my mind to-day than to quote from an eminent New England prelate: "Life is so very short for all of us; shorter still to those of us who are eager to do something that will not perish when we die. The years fly by at a thundering pace which frightens us with its dreadful speed. And when it is over and gone the one thing that will last will be the memory that we have left behind us in the hearts that loved us, in the souls which understood our purposes and which beat in unison with our noblest impulses. That is our only legacy—the only one really worth leaving to history and posterity."

And now, gentlemen, in accordance with the custom of my predecessors, the thirty-fifth annual meeting of the American Laryngological Association is declared open and ready for business. And I thank you for your co-operation in making up the programme and bespeak your active interest to carry it out to a successful termination.

ATROPHIC RHINITIS IN ITS HISTORICAL ETIOLOGICAL AND HISTOLOGICAL ASPECTS.

BY JONATHAN WRIGHT, M.D.

History.

The interest which has arisen among rhinologists to arrive at a better understanding of the lesions of atrophic rhinitis in their correlation with the clinical symptoms is the excuse for my including in this paper an exhausting but not an exhaustive review of its modern history.

The point of view from which I start is to attempt by a process of induction to lay before you and bring into prominence those facts in it which lead to the conclusion that there is some common underlying lesion which binds together the allied clinical phenomena of the different varieties of atrophic rhinitis and to indicate that affiliation with other lesions which points to the same conclusion. That lesion I believe to be a periostitis of the thin scroll bones, a common enough process, but a lesion in a unique situation. Nowhere else in the body have we thin plates of cancellous bone between two layers of fibrous periosteum. Nowhere else in the body have we thin sheets of cartilage between two layers of perichondrium. In hypertrophic rhinitis, or, better in all chronic rhinitis, we have a periostitis. In all sinus affections we have a periostitis. These being the allied local lesions with which we have to do, this being the unique bone configuration with which we are confronted, I turn to the account of the work done on atrophic rhinitis chiefly in modern times. After that, with your permission, I will take up some of the results of my own objective study of the histology of the lesion.

There is no doubt that the history of atrophic rhinitis may be traced back to the remotest antiquity in the history of civilization, especially that variety of it associated with crusts and a foul odor. It seems likely that the reference in the

Talmud to a polyp in the nose, causing a bad smell, to which I have referred elsewhere,[1] is an indication of its occurrence among the early Jews. The Hindu Susruta spoke of it, under the name of cutinaska, as one of the thirty-one diseases of the nose. While in the medicine of the Greeks I have noted no decided indication of the existence of ozoena, as we understand it, there is a reference in Celsus to the fact that the Greeks themselves gave the name of ozoena to the literature of medicine, and he recognized that "it is hardly possible to cure this disease." He recommended its treatment, in some cases, by the application of the actual cautery, but he also employed other methods. Galen recognized it, as indeed did the post-Galenic writers, the Arabians and the physicians of the Renaissance. It may be found spoken of by Fabricius ab Acquapendente, who modified the procedure of Celsus by inserting a metal speculum in the nose and by putting the hot iron in that, which imparted heat to the tissues without actual cauterization. This modification was adopted by later writers.

However, it is impossible always to know just how closely ancient diagnosis and differentiation correspond with our own. That in individual cases it must have been faulty we realize from the fact that in quite modern times we are able to note the absence of a differentiation which has become essential to our present intelligent study of the disease. I therefore hasten on to that period.

In 1866 Weber,[2] who had devoted himself largely to the study of the ear, contributed an article to one of the reference handbooks of surgery on nasal disease, from which it is quite apparent that atrophic rhinitis as we know it—non-syphilitic—without the presence of a foreign body, was undifferentiated at this time.

Zaufal[3] in 1874 claimed that the ozoena present in some cases of catarrh is associated with a congenital deficiency in the development of the inferior turbinated bone. This idea that it is connected with a congenital bone defect was for many

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[1]Wright: History of Laryngology, 1st ed., p. 16.
[2]Weber: v. Pitha and Billroth, Bd. III., 1 abth., 2 heft, p. 177, 1866.
[3]Zaufal: Aertzl. Correspondenzblatt f. Boehmen, No. 23-24, 1874.

years subsequently (1898) expanded in the work of Meisser[4] to the conception that it is due to an abnormal wideness of the nasal fossae, which is congenital. Alexander quotes Sauvages[5] as having claimed in 1763 that it is due to the nose being too narrow. Hopmann[6] again in 1893 had claimed the nasal fossae are congenitally too short. As recently as 1908 he has reiterated[7] his belief in the shortening of the septum as an indication that a congenital conformation of the nasal fossae is the cause of ozoena. Exactly how this condition referred to by Hopmann should have an influence on nasal affections is not very clear, nor is the idea attributed to Sauvages very suggestive, but the too great roominess of the nasal chambers, at first thought, would seem a more plausible explanation, as a greater volume of air contained in them would provide for the evaporation of a greater amount of moisture and so tend to crust formation. But as a matter of fact it soon appears on reflection that this rests upon erroneous conceptions. The air is a moving current, and there can be but little difference in the evaporation coefficient whether the same volume of air passes through a small chamber or a large one. In fact, so far as there is provision for evaporation the advantage lies with the normal nose, as compared to the larger chambers, supposedly attendant on the beginning of the atrophic process, inasmuch as the air current is more closely in contact with the mucosa and can carry away the moisture somewhat more rapidly. As a matter of fact the inception of these ideas rested upon a very insecure basis. Little had been learned of rhinology by observers using modern methods of examination and still less by students of nasal histology. The same may be said of the idea of Michel[8] that atrophic rhinitis is essentially a disease of the accessory sinuses. This is still a prominent feature of discussions on the etiology of accessory sinus dis-

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[4]Meisser: Archiv. fur Laryngologie und Rhinologie, Bd. 8,533, 1898.
[5]Sauvages: Nosalogia Methodica, T. III, 1763.
[6]Hopmann: Archiv. f. Laryngologie und Rhinologie, Bd. 1, heft 1, 1893.
[7]Hopmann: Zeitschrift f. Laryngologie, Rhinologie und ihre Grenzgebiete, Bd. 1, p. 305, 1908.
[8]Michel: Krankheiten der Nasenhoehle und des Nasenrachenraums, Berlin, 1876.

ease. It also rested, especially at that time, upon insufficient differentiation. Indeed, its prominence as an etiological factor in the minds of rhinologists of the present day may be said to be due to an inadequate separation of the various kinds of ozoena from one another, and to the inadequate separation of different kinds of atrophic processes in the nose from one another. Crusts may form from sinus disease as well as from an atrophy of the nasal mucosa. There may be an ozoena and an atrophic process which has extended to the mucosa of the accessory sinuses. Elsewhere I have recently referred to the modern history of the accessory sinus disease in its relationship to atrophic rhinitis,[9] and I will not refer to it here again except incidentally.

There is an atrophy of age and atrophy following nasal operations. As in almost every domain of medicine history here takes account of the differentiation of phenomena in the course of the discussion of theory and the acquisition of our knowledge of facts. Hartmann,[10] in 1878, as a result of post mortem examinations, had already refuted Michel's idea as to the genesis of ozoena in the accessory sinuses. He was inclined to accept Zaufal's theory. Tissier,[11] in 1894, claimed it to be a bone disease of the ethmoid labyrinth in the embryo and the infant. The accessory sinuses are thus affected in his view as a continuation of a congenital condition, the secretions forming a favorable medium for the growth of bacteria. Gerber,[12] in 1900, examined the question of nasal anomalies in their relation to the etiology of ozoena and came to the conclusion that the roominess of the fossae and a depressed nasal arch (chamaeprosopia and platyrrhinia), often combined with syphilis, are predisposing factors. In later years there has been a recrudescence of the belief that ozoena is due to a necrosing ethmoiditis,[13] and again the ethmoid and sphenoid cavities have been vigorously attacked.

[9]Wright: The Laryngoscope, Feb. 1913.

[10]Hartmann: Deutsche Medizinische Wochenschrift, March 30, 1878.

[11]Tissier: Annales de Medecine, January, March, 1893; ibid, November, 1893; Annales des Maladies de l'oreille, etc., No. 10, October, 1894; L'ozene, son unite, ses lesions generatrices, sa traitement, 1894.

[12]Gerber: Archiv. f. Laryngologie, No. 10, p. 119, 1900.

[13]Lavrand: Archives Internationales de laryngologie, T. XXX., No. 2, 1910.

In the early work of B. Fraenkel[14] may be noted the first attempt to separate cases of true ozoena from those of syphilitic ozoena. While he thus erected the typical atrophic rhinitis with ozoena into a separate entity, he failed to indicate that it was dependent also upon a bone change, as is the ozoena of a syphilitic nose. His idea that it was a specific change in the secretions was a natural prelude to the belief which soon arose that this depended upon some specific form of bacterial life. Loewenberg[15] described, in 1885, a bacterium constantly found in the secretions of ozoena, and this has been many times confirmed by other observers since then. Klamman,[16] Thost,[17] Seifert,[18] Strauch,[19] Valentine,[20] Hajek,[21] Reimann,[22] Abel,[23] Struebing,[24] and others contributed to the bacteriology of ozoena within a few years after Loewenberg's paper, but the etiological importance of a bacterium, in spite of much recent literature as to other germs, has not been accepted as preponderating in the causation of atrophic rhinitis, and it cannot be said that bacteriology so far has materially elucidated the mystery of the etiology and pathology of the disease. These writers for the most part looked upon micro-organisms with a mental magnification borrowed from the high contagionist craze of the day. For them the bacterium was the primal factor in the disease. Naturally this left the atrophic bone change even more completely unprovided for than did the subsequent sinus theory of Gruenwald and his followers, to which I have referred as starting with Michel. Fraenkel divided nasal catarrhs into hyperplastic and atrophic, regarding the

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[14]Fraenkel: Ziemssen's Handbuch der Spez. Pathol. und Therap., 1 Aufl. 1876, 1 heft, p. 125.

[15]Loewenberg: Deutsche Med. Woch., Nos. 1, 2, 1885.

[16]Klamman: Allg. Med. Centr. Ztg., 67, 1885.

[17]Thost: Deutsche Med. Woch., No. 10, 1886.

[18]Seifert: Volkmann's Vortraege, No. 240, 1884.

[19]Strauch: Monatsch. f. Ohrenheilk., 6 and 7, 1887.

[20]Valentine: Correspondenzblatt f. Sch. Aertzte, p. 141, 1887.

[21]Hajek: Berl. Klin. Woch., No. 33, 1888.

[22]Reimann: Inaug. Dissert. Wuerzburg, 1888.

[23]Abel: Zeitsch. f. Hygiene, XXI-I., 1895.

[24]Struebing: Muenchener Med. Woch., Nos. 39-40, 1895.

latter as a sequel of the former. He also admitted the influence of various dyscrasiae as etiological factors. More than thirty years later he had the satisfaction of reminding[25] his colleagues that this early classification is fundamentally to-day the basis of differentiation of intranasal inflammations. In 1879 Eugen Fraenkel[26] studied sections of the atrophic nasal mucosa under the microscope and noted changes in the epithelium, though he asserted that it is destroyed and replaced by other cells. He remarked on the few acinous glands to be observed. He believed there was always an underlying dyscrasia of tuberculosis or of syphilis. He believed there were always deep lesions of the mucosa and of the bone. In the same year Gottstein,[27] though believing in its systemic nature, recognized ozoena as a constant symptom of that stage of a chronic rhinitis in which it has come to an atrophy of the mucosa and the glands in it. The vitiation of the secretions from this cause he proposed to treat by the use of intranasal tampons of cotton left in the nose for twenty-four hours or more. Hermann Krause,[28] in 1881, described microscopic appearances in two cases of ozoena and noted the presence of evidences of fat and fatty acids in the secretions and in the mucosa, fibrosis of the stroma and blood vessels and the relative increase of lymphoid cells. The destruction of glands he believed to be due to limitation of the blood supply and the fibrosis of the blood vessels. These ideas were, many of them, introduced in America by Bosworth in several excellent papers[29] in 1882 and in his Treatise of the Diseases of the Nose and Throat. He believed atrophic rhinitis with ozoena was usually the sequence of a purulent rhinitis in childhood. Bruegelmann,[30] in 1884, looked upon the nature of ozoena as a suppuration of the nasal turbinated bones. He

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[25]Fraenkel: Berliner Laryngologische Gesellschaft, ref. Semon's Centralblatt f. Laryngologie, 1907, p. 115.

[26]Eugen Fraenkel: Virchow's Archiv., Bd. 75, p. 45, 1879.

[27]Gottstein: Berliner Klinische Wochenschrift, No. 37, Sept. 16, 1878, p. 554; Breslauer Aertzliche Zeitschrift, No. 17, 1879, p. 169.

[28]Krause: Virchow's Archiv., Bd. 85, 1881.

[29]Bosworth: Transactions International Medical Congress, 1881, III., p. 329; New York Medical Record, XXI., No. 23, June 10, 1882; Archives f. Laryngologie, Vol. III., 1882, p. 232.

[30]Bruegelmann: Monatschrift f. Ohrenheilkunde, No. 5, 1884.

thought the increased dimensions of the nasal chambers an anomaly of development, and he believed that this and a scrofulous diathesis are predisposing factors in the etiology.

Zuckerkandl brought the discussion to its more useful channels in refuting the fallacies of the theories dependent on a congenital bone defect or abnormality, and in suggesting that the changes in the bone are apparently the result of an intravitam inflammatory process—but it was a number of years before this view became the prevailing one. Many papers were written in support of the view that it is due to a congenital conformation of the nasal fossae. These are reviewed in the paper of Meisser (l. c.) and the later one of Alexander. The paper of Cholewa and Cordes[31] is to be mentioned as claiming that the essential lesion is a bone lesion, but a peculiar one, not of an inflammatory nature. As for the changes observed in the mucous membrane itself, these have been variously described as primary and secondary to a bone lesion by numerous observers, among whom we may name Habermann[32] in 1886, Schoenemann[33] in 1902, Oppikofer[34] in 1907 as representative of opinions expressed at isolated times. The whole question is exhaustively reviewed by Alexander[35].

John N. Mackenzie[36] in 1884 expressed the belief that there is always a hypertrophic stage to atrophic rhinitis. Moure[37] declared himself a sharer in the belief of Gottstein that the essential lesion in atrophic rhinitis lies in the glands. Habermann (l. c.) also accepted this view. The involvement of the serous and mucous glands is well established in present day nasal histology, but it has never been generally accepted

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[31]Cholewa and Cordes: Archiv. f. Laryngologie und Rhinologie, Bd. VIII, p. 49, 1898.

[32]Habermann: Zeitschrift f. Heilkunde, Bd. VII, 1886.

[33]Schoenemann: Virchow's Archiv., Bd. 168, 1902.

[34]Oppikofer: Archiv. f. Laryngologie und Rhinologie, Bd. XIX, p. 65, 1907.

[35]Alexander: Ibid., XXII, p. 260, 1909.

[36]Mackenzie: Phila. Medical News, No. 14, Oct. 4, 1884, and April 4, 1885.

[37]Moure: Compte rendu de la Societe francaise d' otologie et de laryngologie, 1885.

as a satisfactory exposition of the whole process or of its etiology.

Luc[38] and Bronner[39] in 1887-1888 again drew attention to the apparent association of ozoena and accessory sinus disease. Gruenwald[40] advocated this idea in his book on nasal suppuration, and that other lesions also were the cause of it, such as adenoid vegetation, etc. Luc[41] in 1881 reported three cases of tracheal ozoena, and a discussion as to priority in the observation of the affection arose with Massei. Zarniko[42] and others asserted that tracheal ozoena may exist without a nasal lesion. Potiquet[43] contributed a number of papers to the earlier discussions of atrophic rhinitis by modern rhinologists.

I have already referred to the literature of the microbian theories of the etiology of ozoena. The similarity of the bacillus found by Loewenberg, Abel and Paulsen in ozenatous cases with the bacillus of Frisch, and both with the bacillus of Friedlaender led to much discussion as to the connection of ozoena, scleroma and pneumonia[44]. While most observers confirmed the early reports of various bacterial forms found in the secretions of ozoena, no one was able to demonstrate the presence of any of them in the subepithelial tissue of the mucosa itself and some of the reputed specific forms were found in normal noses or in noses not the site of atrophic rhinitis. Perez[45] not only discovered a cocco-bacillus as the cause of ozoena, but found it also in a dog, and, injected into rabbits, it caused gradual atrophy of the turbinated bones. He suggests that it is acquired by man from the dog. Hajek[46] who made investigations on the bacterial contents of

[38]Luc: Societe de Medecine pratique, Feb. 1887.

[39]Bronner: British Medical Journal, March 3, 1888.

[40]Gruenwald: Muenchener Medizinische Wochenschrift, Nos. 42-43, 1893.

[41]Luc: Archiv. de Laryngologie, etc., Feb. 15, 1888, April 15, 1888.

[42]Zarniko: Deutsche Medizinische Wochenschrift, No. 29, 1898.

[43]Potiquet: Revue hebdomadaire de laryngologie, otologie, rhinologie, Jan. 1, 1890.

[44]Struebing: l. c.; Abel: l. c.

[45]Perez: Centralblatt f. Laryngologie, 1909, p. 98.

[46]Hajek: Anzeiger, No. 28, 1887. (Gesellschaft der Aertzte in Wien.)

the nose, expressed his belief that the organisms found in ozenatous cases had little or nothing to do with the etiology of the disease. He believed that atrophic rhinitis had always a preliminary stage of hypertrophy. In the discussion on his paper in 1887 in which he asserted this, and that the Friedlaender bacillus often found in the nose played a secondary part even in pneumonia, he was opposed by Weichselbaum, Paltauf and Roth.

Siebenmann[47], who was a collaborator with Meisser in the examination of cranial form as a factor in the etiology of ozoena, made a careful study of the histology of atrophic rhinitis, but he, as many other writers, laid a stress upon the specificity of the metaplasia of the epithelium of the surface which would not have been the case if such work had been controlled sufficiently by the examination of this element in normal noses and in other pathological conditions, since in adult life there is always much of it to be noted. Cozzolino[48] believed the lesion first begins as a keratosis of the surface epithelium and becomes secondarily bone disease. There was a tendency with many others, as with Rethi[49], for example, to consider the ozoena as something specific and aside from the anatomical lesions of atrophic rhinitis.

I have noted that from time to time authors had incidentally remarked on the connection of tuberculosis and ozoena, owing to their having a common basis in malnutrition. Theisen[50] in 1904 made it the subject of a paper. Broeckaert[51] contributed to the study of its histology the expression of his opinion that the disease was due to a toxin produced by a parasyphilitic or a paratuberculous systemic infection. These terms came into use about this time, and their exact pathological significance is still enshrouded in mystery. Frese[52] was of the opinion

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[47]Siebenmann: Correspondenzblatt f. Schweizer Aertzl., H. 5, 1900.

[48]Cozzolino: Annales des Maladies de l'oreille, etc., April, 1894.

[49]Rethi: Archiv. f. Laryngologie und Rhinologie, II, 2, 1894.

[50]Theisen: Transactions American Laryngological Association, 1904,

[51]Broeckaert: La Presse oto-laryngologie Belge, No. 7, 1905; Ref.: Centralblatt f. Laryngologie, 1906, p. 273.

[52]Frese: Archiv. f. Laryngologie und Rhinologie, Bd. 20, p. 459, 1908.

that most of the cases are of syphilitic origin. Sobernheim[53] in 1909 reported that seventeen cases of typical ozoena gave a negative Wassermann reaction, adding another proof to the view that the nasal affection may exist without any syphilitic antecedent. A number of others testified to the same effect. It was spoken of as a larval tuberculosis.[54] Schoenemann[55] in 1909 traced a connection between the occurrence of eczema and that of ozoena. From time to time various authors have suggested that ozoena is a trophoneurosis.[56]

Treitel[57] made an attempt to determine the age at which ozoena first shows itself and concluded that in the great majority of cases it was in the school age of children that it developed—4 to 14 years. In all treatises on the subject since the affection was first differentiated by B. Fraenkel the preponderance of the female sex in the cases affected by it was noted.

The treatment of atrophic rhinitis, especially of its chief symptoms, ozoena, has been varied. Going back to the time of its differentiation, we have already noted that tampons were recommended by Gottstein, and in the earlier history of rhinology this was a common form of treatment for all nasal affections, the linen or other absorbent tampon being saturated with various medicaments. Cauterization, we have seen, was also recommended by the ancients for what they called ozoena, and in 1884-5 it was recommended[58] again as a thera-

[53]Sobernheim: Centralblatt f. Laryngologie, 1909, p. 318; Archiv. f. Laryngologie und Rhinologie, Bd. 22, heft 1, 1909.

[54]Caboche: Annales des Maladies de l'oreille, etc., Sept., 1907.

[55]Schoenemann: Verhandlungen des Vereins deutscher Laryngologen, 1909, p. 168.

[56]Jouty: Archives Internationales de laryngologie, T. XXIX, No. 3, 1909.

Baumgarten: Archiv. f. Laryngologie und Rhinologie, No. XXII, p. 492, 1909.

Lavrand: Archives de laryngologie, etc., XXX, 1910, p. 400.

[57]Treitel: Archiv. f. Laryngologie und Rhinologie, Bd. 16, p. 336, 1904.

[58]Garrigou-Desarenes and Mercie: Du catarrhe chronique des fosses nasales et de l'ozene; traitement par la galvano caustique chimique; Abeille: Courrier Medicale, XXXV, p. 448, 1885. Ref. Centralblatt f. Laryngologie und Rhinologie, I, p. 319; III, p. 6.

peutic agent in its modern differentiation. In most of the writers on the subject recommendation for treatment included thorough cleansing and the application subsequently of some stimulating medicament, like thymol, aristol, turpentine, ichthyol, etc. Braun, Laker and Demme[59] recommended (1891) vibratory massage. This was employed by others either by hand or by electrically driven machinery. Faradism was also used,[60] also the electrolytic application of chloride of copper[61] and submucous electrolysis.[62] Dionisio[63] employed various forms of phototherapy in ozoena, and in the same year (1903) Casassa[64] reported on the therapeutical use of radium.

Brindel[65] in 1902 inaugurated the treatment of ozoena by the injection of paraffin under the atrophied mucosa, and this method of treatment has persisted to the present time, it being strongly recommended by Hutter[66] ten years later. Broeckaert,[67] who adopted the idea promptly, subsequently supplemented it by more vigorous measures. It was practised by Fliess[68] and by many other rhinologists for a time

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[59]Braun, Laker and Demme: Berliner Laryngologische Gesellschaft, 17 July, 1891; Berliner Klinische Wochenschrift, No. 46, 1891.

For mechanical devices for vibratory massage of the mucosa for this and other affections see: Daae: Archiv. f. Laryngologie und Rhinologie, II, 2, 1894, p. 265; Jankau: Monatschrift f. Ohrenheilkunde, No. 5, 1897.

[60]Garrison: Journal Ophthalmology, Otology and Laryngology, Oct. 1893.

[61]Jouslain: Societe d'electrotherapie, 21 April, 1892.

[62]Cheval: Revue de laryngologie, Aug. 11, 1895; Bayer: Muenchener Medizinische Wochenschrift, Nos. 32-33, 1896. Revue hebdomadaire de laryngologie, No. 22, 1896.

[63]Dionisio: Giornale del R. Academia di Medicina di Torino, Jan., 1902; July, August, 1903.

[64]Casassa: Archivii Ital. de Otologia, Rinologia e Laringologia, August, 1903.

[65]Brindel: Presse Medicale, June, 1902.

[66]Hutter: Archiv. f. Laryngologie und Rhinologie, 1911, Bd. 24, p. 189.

[67]Broeckaert: La Belgique Medecine, Nos. 42-43, 1903; Annales des Maladies de l'oreille, etc., VII, p. 59, 1903.

[68]Fliess: Berliner Laryngologische Gesellschaft, Nov. 13, 1903. Ref.: Centralblatt f. Laryngologie und Rhinologie, 1904, p. 255

after this, until the report of an embolus of the retinal artery in two or three cases of the injection of paraffin in the skin of the nose discouraged for a while frequent resort to it. On the strength of his belief that atrophic rhinitis with ozoena was the result of a disease of those cavities, Broeckaert.[69] in 1905, in addition to the use of paraffin, proposed to operate radically on the ethmoidal sinuses and to curette the sphenoid. He removed the middle turbinate, allowing the inferior to remain, reinforcing it with paraffin injections, and injections of it also into the septal walls and floor of the nose. He proposed also to destroy the naso-sinusal wall of the antrum, but not to curette the latter unless it was diseased. He claimed the injection of paraffin was efficient not only as a prothesis, filling out the large spaces, but as a stimulant, modifying the nutrition of the mucosa. Guyot[70] employed suction of the nasal chambers by mechanical methods in the treatment of ozoena. A number of cases were treated with injection of anti-diphtheritic serum because diphtheroid bacilli were found in the crusts—chiefly by Italians.[71] Skillern and Holmes[72] used vaccines made from the pure culture of the Abel bacillus. Emulsions of the culture of the lactic acid bacillus have been used by many in the treatment of ozoena.[73] Foy[74] in 1911 claimed considerable benefit can be attained by the patients being taught to breathe persistently and properly through the nose. Ozoena has been treated with hot air, dry or moist.[75]

In the January, 1912, number of Semon's Centralblatt fur Laryngologie, etc., there is a very valuable review of the literature of ozoena during the years 1909, 1910, 1911, by

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[69]Broeckaert: La Presse oto-laryngologie Belge, No. 7, 1905, No. 5, 1906; Belg. oto-laryngologische Gesellschaft, Dec. 10, 1905. Ref.: Centralblatt f. Laryngologie, 1906, p. 273.

[70]Guyot: La Clinique, No. 24, 1906.

[71]Belfanti and della Vedova, Gradenigo, Arslan and Caterina: Archivii d'Otologia, Nos. 2-3, 1896; Compaired: Annales des Maladies de l'oreille, etc., No. 5, 1897; Molinie: Annales des Maladies de l'oreille, etc., No. 4, 1899.

[72]Skillern and Holmes: New York Medical Journal, August 15, 1908.

[73]Stepinski: Archives Internationales de laryngologie, T. XXXI, No. 1, 1909.

[74]Foy: Annales des Maladies de l'oreille, T. XXXVI, No. 12, 1911.

[75]Thost: International Medical Congress Transactions, Bd. II, p. 29; Moller: Verhandlungen des Vereins deutscher Laryngologen, 1909-10

Alexander. Among the points to which he gives prominence is the inequality in the distribution of cases geographically, racially and socially, its incidence as to sex and age. He drew attention to the fact that while the Wassermann reaction in his own experience and in that of others had given negative results in cases of ozoena, he had himself seen a typical case of ozoena, the offspring of a syphilitic father, the sister of a congenitally syphilitic brother, and in her case the Wassermann reaction was also negative. He also drew attention to the fact that while ozenatous subjects rarely contract syphilis, such cases have been reported by Fraenkel and Sobernheim (l. c.) and others, and that the question of the relationship of true ozoena to syphilis is not by any means settled. It may be remarked that the subject is much embarrassed by the confusion which still reigns as to the clinical differentiation of the cases. With syphilitic ozoena on the one hand and atrophic rhinitis on the other, without crusts or odor, it is difficult for an author to keep constantly in view the typical cases of atrophic rhinitis with ozoena. The confusion is increased by the introduction of the consideration of accessory sinus disease attended by intranasal crust formation. In this excellent review it can be plainly seen that with the exception of the therapy, which is always new, optimistic and futile, there has been scarcely a question raised in the literature of the subject during the three year period with which it is concerned that had not been thoroughly discussed in the former three decades in discussions of the phenomena presented by ozoena.

Etiology.

Most modern observers, who have devoted themselves to the histology of the lesions of atrophic rhinitis, believe it is primarily a bone disease. In the consideration of the etiology various predisposing factors have been urged, some of them with considerable plausibility, others with none. General systemic malnutrition in children, sometimes dependent on syphilis, seems warranted from the number of cases observ-

able in this class, often seen in two or three children of the same parents. Sex quite evidently is a determining factor, as a very large majority of the cases occur in females. Puberty also is clinically observed to have a determining influence on the advent of the symptoms.

These factors are observable in the apparent etiology of those cases of chronic rhinitis with crust formation and the typical ozoenic odor. In regard to the latter, while we speak of a typical ozoenic odor it seems probable, from the state of opinion as to the bacteriology of the disease, that there is no specificity about it. Large numbers of bacterial forms, swarming in a lipo-proteid excretion of the mucous surfaces, set up chemical changes in which the volatile and non-volatile fat derivatives are concerned, which give rise to the odor.

While space forbids a discussion of the many theories advanced in regard to the etiology, it may be well to say that syphilis, long regarded as an etiological factor from the point of view of heredity, has recently been investigated in its relationship to ozoena by the Wassermann reaction. Negative results were almost uniformly obtained when complement fixation was tested for in the serum of the blood of ozoenatous subjects. How far this can be regarded as conclusive in negation of the idea of a syphilitic hereditary factor in the etiology of true ozoena it is too early yet to decide.

Excluding the cases of active tertiary nasal syphilis, as we do in modern rhinology in speaking of ozoena, there is little warrant for the belief that hereditary syphilis has more to do with the etiology of atrophic rhinitis than that the syphilitic dyscrasia, like the tuberculous dyscrasia, is the cause of a lowered state of general vitality. Like tuberculosis, atrophic rhinitis is the disease of poverty. It occurs in the well-to-do just as phthisis does, but it is a disease of malnutrition, the usual cause of which, as we know, is poverty.

There is an affiliation of atrophic rhinitis with accessory sinus disease by virtue of the fact that the process itself occasionally extends to the mucosa of these cavities. There are also certain cases in which a chronic discharge from the

posterior ethmoidal and sphenoidal sinuses exists with crust formation. In the former category of cases surgical treatment of the sinus accomplishes nothing, in the latter category of cases eradication of the sinus affection cures the ozoena. In the majority of cases of atrophic rhinitis with ozoena the sinuses are not involved in any way, even in advanced stages. Frequently the openings of the sphenoidal sinus can be perceived by anterior rhinoscopy, owing to advanced atrophy, and the mucous membrane may be seen to be free of crust or other discharge.

Atrophic rhinitis sometimes exists in one nostril, when there is marked deviation of the septum. The concavity on the one side shows abundant crusts and an atrophic mucosa. Straightening of the septum regularly cures the condition. Atrophic rhinitis not infrequently follows extensive operative destruction of intranasal structure. These undisputed phenomena lend some plausibility to the theories and observations advancing, as an etiological predisposing factor, the congenital roominess of the nasal chambers. The arguments and facts usually adduced in support of such theories have not sufficed to convince the majority of rhinological observers of their tenability. Finally a certain amount of atrophy, without crusts, is the physiological accompaniment of old age. There is plainly a shrinking of both bone and mucosa.

There is hardly any histological lesion which could serve as a common cause for changes in the mucosa of all these cases except that of a bone lesion which has interfered with the nutrition of the soft parts. The bone itself is affected in them all, and while there is no satisfactory evidence in the bone itself of inflammatory action, the process is manifestly associated with a pre-existing chronic inflammation of the mucosa in a sufficient number to suppose that in these at least the bone atrophy is due to it. An analagous bone lesion may be supposed to exist in the other cases. It must be confessed, after all is said, the etiology and even the succession of histological changes is far from satisfactorily understood or accurately known.

Pathology.

In this form of nasal inflammation the metaplasia is more marked than in the hypertrophic form and it is constantly present. Even in fairly normal nasal chambers the cylindrical epithelium is in places frequently replaced by flattened cells. In atrophic rhinitis this is universal. Yet, so far as I know, there is no stickle cell formation such as is sometimes seen in the epithelial metaplasia of other morbid processes. There is a tendency to cornification of the surface layers.

There is beneath the epithelium an infiltration of the stroma with a relatively much increased number of round cells. These are marked around the ducts of the acinous glands. There is a steady encroachment on the epithelial structures of the glands by these round cells and a lowly organized fibrous connective tissue which finally all but destroys all traces of glandular structure. The same may be said for the blood vessels. Whether cavernous sinuses in these cases have ever been much developed at puberty or not is a question. At any rate, the blood vessel walls have been to a large extent obliterated by the fibrosis and the round cell infiltration. That in this process of cell degeneration there is the production of considerable fatty detritus is probably true, but to the extent one would infer from the accounts of some observers is certainly not my experience. This proteid decomposition evidently finds its way to the surface with the copious discharge of serum which takes the place of the normal viscid fluid of the glands, and there its rapid evaporation leaves behind in the form of crusts a lipoid proteid precipitate which rapidly ferments and is split up into fatty acids by various bacteria. A large number of these have been found and several have been described as specific in the disease, but neither the bacilli of Loewenberg, Perez, Pes-Gradenigo, nor the diphtheroid bacillus, all of which have been urged as etiological factors, have been accepted as of any significance in the causation of the lesions. It seems probable that in the sense that there is an antecedent hyperplasia of the periosteum in atrophic rhinitis, we may say that hypertrophy is followed by atrophy in certain cases of nasal inflammation, but it seems quite certain that while this pre-

existing periosteal hyperplasia may be present in the larger number of cases of typical atrophic rhinitis with ozoena, this is the only element in the mucosa which is affected by preliminary hyperplasia. That this is always affected primarily must also be acknowledged as more or less hypothetical. Nevertheless, the most significant change in structure is the thickening of the periosteal layer. Inasmuch as the distribution of nutrient arteries to bone and to mucosa lie in this element of the mucosa, inasmuch as it seems to be a fibrosis which ultimately destroys the glands and the blood-vessels of the mucosa itself, it is a fair conjecture that it is in the periosteum and the perichondrium, for the septal cartilage also becomes thin and even in places absorbed, we must seek for the root of the changes which give this nasal disease unique characteristics.

The histological features of the disease seem to justify us in calling it a fibrosis, but the clinical features which accompany them justify us rather in calling it a mystery. There is no analogue to it elsewhere in the body. This gives us a clue to its essential nature. There is no other region in the body where we have a very thin plate of bone covered on both sides by a vascular secreting mucous membrane and separated from the bone on each side by a thin layer of periosteum which carry its nutrient blood-vessels. Often present in early childhood, associated with puberty in women, the symptoms ceasing for the most part after middle life, not beginning—except in the rarest cases—after adolescence, apparently not necessarily preceded by a condition of chronic inflammation or by hypertrophy, conceiving of it as initially a fibrosis of the layers of connective tissue next the bone and cartilage is a matter of some difficulty, yet after all that seems the most reasonable hypothesis purely from a histological standpoint.

In order to understand the essential lesions of the stroma of mucous membrane in atrophic rhinitis, I must repeat somewhat the remarks I have made elsewhere in a recent series[76] of articles on the contractile elements in the connective tissue:

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[76]New York Medical Journal, 1910.

Looking for the first time at the picture presented by sections of the nasal mucosa of animals, stained by the Weigert formula or one of its modifications, the observer is struck with the abundance of elastic fibres. Their profusion at first seems not to be more pronounced, on the average, in one locality than in another, but it soon becomes evident that, aside from their great abundance in the walls of the large blood-vessels, the most constant and the most richly supplied locality is the periosteum and the deeper layers of the stroma intimately associated with it. Thick, interlacing bundles are always demonstrable, running, in an irregular way, parallel with the planes of the bone. Everywhere the blue-black threads run through the middle layers of the stroma, always either parallel or obliquely to the surface. Nearly always demonstrable is a special layer of fibres situated in the stroma just under the epithelium at varying distances from it, but in such relation to it and in such arrangement that one can not fail to perceive that it subserves a special physiological need, somewhat apart from the general tonicity the widely scattered fibres elsewhere would give the mucosa.

The persistence of this layer of wavy fibres just under the epithelial line is not infrequently noted in a mucosa considerably altered by hypertrophy. It is a layer indefinite in extent and somewhat inconstantly seen in the normal mucosa in the human infant and in animals. It is well to draw attention to the fact that never in the free stroma do the elastic fibres run in planes vertical to the surface or vertical to the periosteum, but always obliquely to them, except for the subepithelial fibres just spoken of, and the elastic fibres of the periosteum itself, which run in more or less horizontal directions in relation to the surface and the bone.

Recently an article by Duerck[77] has drawn attention to straight telegraph wirelike fibres revealed in connective tissue generally and in arterial walls by some of the Weigert stains for nerve sheath demonstration. He seems to think these had not been previously demonstrated in the walls of the arteries. While they may not have been especially emphasized in the

[77]Virchow's Archiv. 189, Hft. I, p. 62, 1907.

work of Koelliker and Ebner, from which he quotes, they certainly are a more or less familiar phenomenon to histologists and may be demonstrated with the haemotoxylon-resorcin stain as modified by Hart. It is not unusual to see these fibres, or fibres at least morphologically similar to them, in the connective tissue of turbinated bodies of infants, and they are seen also in that of the monkey.

Breugel[78], who used the "haemotoxylon eisenlack" stain for sections of the penis, also demonstrated them there. We may imagine they furnish in the walls of the arteries and elsewhere that form of elasticity lent to a lady's corset by the whalebone stays, but it is not at all certain that their straightness, their "telegraph wire" appearance may not be due to their being ordinary elastic wavy fibres stretched out straight by traction or by virtue of their position.

The distribution of elastic tissue around the gland acini is not very marked, or apparently in such a mechanical arrangement as to work in the most efficient manner in expelling the contents. While it is true that in some cases it is demonstrable in the material from animals, such is not uniformly the case. One gains the impression that such expulsion of mucus from the glands as may occur from the contractile action of the tissues, arises more from the general elasticity of the stroma than from a special network around the glands. Although the description of Schiefferdecker[79] is not very accurate, it seems to me, I repeat what he says. Backward from the vestibuli nasi "with the beginning of the glands, the elastic fibres, especially in the deeper parts of the stroma, become more slender, but are nevertheless still thick; they surround the glands and, singular to say, reach a greater thickness exactly at those places which lie between the epithelium and the glands, so that this part appears as the characteristic continu-

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[78]Virchow's Archiv, 194, Hft. 2, 1908, p. 255.

[79]Heymann's Handbuch der Laryngologie und Rhinologie, III, erste Haelfte, p. 95

ation of the network hitherto penetrating the whole stroma."[80] Now while this may be the character of the elastic fibre arrangement bordering on the vestibule, in the deeper portions of the nose such is not the case. The same author subsequently speaks of the glands of the deeper parts of the nose being surrounded by a membrana propria. It is true as will subsequently appear in a discussion of pathological conditions, that one can observe appearances, as if the base of the gland cells exuded a homogeneous substance which forms a sort of an incomplete and irregular hull to a gland acinus; but it is very difficult to be sure that this, in the material from animals, is not simply a slightly more dense arrangement of connective tissue fibrils. Among these, as I have said, may frequently be traced the elastic fibre of the stroma, but there is no specific periglandular network of fibres taking the Weigert stain, nor is there anything which can be called a membrana propria.

In the walls of the nasal blood-vessels I am not aware there is any very exceptional feature to the distribution of the elastic fibre. In the endothelium of the single walled capillary the occasional appearance of a purple stain may be thought to argue for an elasticity which is more evident, judging from the stains, as the thickness of the walls increases. The intima of the venous sinuses is very thick, and the longitudinal elastic fibres always stand out as sharp landmarks in sections of the erectile tissue colored with Weigert's stain. It also mingles freely with the external coats of the venous sinuses.

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[80] Schiefferdecker has had the same experience with the apparent capriciousness of elastic tissue stains, and he also is not disposed to trust the result of tinctorial reactions. It seems to me we are justified in supposing that there are variations of chemical and tinctorial reactions which do not influence elasticity, but it is also possible that a functional demand may temporarily effect a chemical, and hence a tinctorial change and also a change in elasticity. In other words, elasticity being an expression of energy, it may vary in the tissues, just as muscular energy varies without betraying itself in structure to our powers of vision. This falls in very well with a recent remark of von Hansemann (Deszendenz und Pathologie, Berlin, 1909), whose ideas on the mutability of tissue-form for more than a decade have been known under the name of "anaplasia." "In man slightly differentiated connective tissue is extraordinarily variable in its occurrence, and everywhere adapts itself in form and position to temporary demands made by normal and pathological states."

The arterial vessels present no especial feature in this regard.

What I wish to dwell on especially is the condition of the elastic fibre itself in the presumably normal material, as distinguished from the elastic fibre and the elastin noted in the material from the nasal mucosa of adults removed in the course of clinical work. Normally the elastic fibre is indefinitely long, slender, branching, usually with finely drawn out ends interlacing everywhere with other fibres. Just where and how they are anchored to other cells is not very clear. The transverse measurement of the elastic fibre varies greatly in different localities. In the nasal mucosa it is of smaller calibre, as seen in the infant and in the monkey. It ranges from a hardly perceptible line, as viewed with the o. im. 1-12 obj., up to perhaps two microns. Koelliker, speaking of the elastic fibre in general, says it ranges up to six microns in diameter, or even to twice or thrice that. In the nose when it exceeds two or three microns it is usually associated in the loose stroma with pathological conditions and itself presents various departures from what I conceive is its normal condition. In my notes I have spoken of this normal condition as "efficient looking," meaning thereby they were arranged in such a way as, supposing they were capable of contraction, their shortening would lead to a condensation of the tissues in which they lie. This was to distinguish them from swollen fibres with abrupt ends, broken into sections, granular, sometimes betraying their former state of continuity only by a row of granules into which they had disintegrated in the course of disease—perhaps at times they break up into this state of discontinuity as a result of the technique of fixation, hardening and staining. Great clouds of elastin granules are thus seen crowding some fields. In the normal mucosa when such clouds exist they are more or less homogenous, the granules for the most part not being resolved by the highest powers of the microscope.

So far as my own convictions go, there can scarcely be a doubt of the origin of the elastin and the elastic fibre as an exudate of the cells in adult tissue, even when the fibre is efficient looking. In normal tissue it is organized into a fibre by

a precisely working machine, but when the stimulus is over great, as in hypertrophic conditions, the fibres are thick and swollen, with abrupt ends, often broken into sections or a row of granules, but frequently existing as a cloud of coarsely granular material in the stroma. Such granules are seen inside of cells, especially at the free surface of gland cells. Up to a certain point this disorderly condition of the elastin corresponds to the extent and the degree of the hypertrophy, and then a change sets in.

I can not here enter into an exhaustive discussion of all the changes in the nasal mucosa supervening upon an hypertrophic rhinitis. I must, however, trace in outline the course of events which can be gathered only from a composite mental impression of the moving picture of morbid processes after years of observation. The static conditions conventionally looked upon as the "pathology" of disease is a sterile way of studying vital problems. Morbid tissue, like normal tissue, is a living thing, moving along the path in which it is guided by the laws of living matter and by, in this locality, the preponderating influence of the environment. At hardly any given moment is the condition exactly what it subsequently becomes. The only constant thing is change. I am compelled then, in attempting to place the changes in the so-called elastin in the proper light, to be a little discursive. There is a growing tendency, for which we must be profoundly grateful, to treat of pathology not as something dead and immovable, but as of something living and mobile, a process and not a state. In chronic inflammation of the nasal mucosa the static conception of it is entirely unintelligible. We must conceive of it as a process in which changes are slowly advancing as the result of age, heredity and environment.

There may be changes in the nasal mucosa as the result of a chronic rhinitis which has not perceptibly increased or diminished the bulk of the tissue. Hypertrophy and atrophy are gross and inexact terms, but they are so fixed in rhinological usage that it is impossible—at least inconvenient—to break away from them. So, notwithstanding the protest I make, in what I have to say it will be more convenient to start from a basis furnished by some of the conventional

divisions of atrophic rhinitis, of no value in themselves except as indicating some of the causes underlying the process.

It often happens that atrophy supervenes upon states of the mucosa whose condition of chronic inflammation has caused symptoms so trifling as not to betray it. It is occasionally the case that oedematous polypi (especially in asthmatic and hay fever cases) spring from a mucosa in which previous inflammatory changes have been insignificant, and the same may be said of atrophic rhinitis. Nevertheless it is true that a periosteal lesion without any antecedent hypertrophy of the mucosa may be the first step in the pathological process which leads to an atrophic rhinitis. Such is a fact, I believe, in those ozoenic cases, not too common now, that a keener differentiation of diagnosis is exercised, which we recognize as having begun in early childhood, when there may have been a half suppressed syphilitic inheritance, or some other systemic deep-lying vice. This also frequently applies to those cases whose symptoms of ozoena, with the characteristic crusts and odor, date back to puberty only. There is another class of cases—pseudo-ozoenic we may call them—which have foul, decomposing crusts without the specific odor, and associated with sinus suppuration. In both these categories there is an antecedent periosteal lesion. In the first, the true ozoenic cases, there is a periosteal lesion due to some unknown systemic cause which has blocked the nutrient arteries of the superjacent soft parts. Such is also the condition *mutatis mutandis* in the other category just mentioned. The suppurative inflammation of the mucosa of the sinuses has spread through the periosteum to the bone. One bone change, we may say, is endogenic, the other is exogenic, and the results (true ozoena and pseudo-ozoena) may be differentiated by careful clinical observation. There is still a third class of atrophy in addition to that succeeding hypertrophy. I refer to the atrophy of age. The bone shrinks, the epithelium is atypical, the venous sinuses are fewer and smaller, the racemose mucous glands are suppressed, while from the single tubed serous glands of Bowman in the upper regions of the nose and from direct transudation a watery fluid flows and drips, often so inopportunely, from the nose of age.

There is always some oedema or serous infiltration accompanying atrophy even in the very last stages. In the end, the connective tissue cells have taken on an embryonic type with branching processes, and such mesh of fibrils as still exist is crowded with the small leucocytes and their naked nuclei. At this stage the elastic tissue in the walls of the radical arteries and veins, even that of the intima, has become disorganized and partially absorbed. The glands, all but perhaps some traces of their ducts, have disppeared and the stroma of the turbinates is little better than granulation tissue.

To summarize, then: In the newborn infant the elastin material exists in the nasal mucosa, so far as selective stains reveal its presence, in the form of a fine network of fibres, and no coarse granular material or swollen fibres. Later in life the latter appear and in hypertrophy are prominent in the landscape of the sections. As changes go on to atrophy of any kind or to oedema the useless non-efficient material is absorbed first and the fine fibres last, perhaps themselves passing through a stage of disorganization. I think there can be no doubt that in adult tissue the granular elastin has an intracellular origin, and it is probable that even some of it thrown out in inflammation may be organized into efficient fibres; but for the most part this attempt at organization is incomplete and imperfect. Like the callus thrown out from bone, it must be looked upon as a process of repair and regeneration. The material carries with it imperfectly the heredity of early embryonic life—the tendency to tissue formation. It is quite impossible to account for the appearances on the supposition that all the disorganized material has had its origin in the degeneration of pre-existing normal fibrils.

In taking up the question of the presence of the smooth muscle cell in the stroma of the nasal mucosa, so interesting from many points of view of a general nature, the special historical questions concerning it have hitherto aroused interest only in one respect. Is it a fact that separate smooth muscle cells or limited bundles of cells lie free in the loose stroma of the erectile tissue of the turbinate bodies, or are they always closely connected with the coats of the large

blood vessels? Zuckerkland[81] and Herzfeld[82] maintain the view, and their position may be regarded as summed up in the words of Herzfeld: "The erectile tissue of the nasal mucosa is rich in organic musculature. This, however, does not lie free in the connective tissue of the erectile bodies, but forms, as in all other parts of the body, the middle tunic of the arteries and veins, and lies closely pressed around the walls of the lacunae of the erectile tissue."

Students of rhinology became familiar with the view of Zuckerkandl, which is of the same tenor, many years ago. While muscle cells had been described in the stroma of erectile tissue elsewhere, the *corpus cavernosum* of the penis, the nipples, etc., the scalp, the skin of many animals, for many years no one ventured to assert in the face of Zuckerkandl's authority their presence in the stroma of the nasal erectile bodies. I had been indisposed to accept the views of Zuckerkandl chiefly for physiological reasons and from the clinical study of the nasal mucosa. Owing to the difficulties of differentiation and the unsatisfactory technique in vogue before Mallory's recent introduction of his connective tissue, stains have added so much to the more exact study of cellular relations it was difficult to acquire sufficient assurance to refuse an assent to an authority so weighty as that of Zuckerkandl. It is true that Schiefferdecker[83] has asserted of late years that muscle fibres do lie free in the stroma, but the confirmation of the assertion was a difficult matter, and it still remains a disputable matter. The difficulties are not wholly technical nor entirely those of personal opinion, but in the question are involved some fundamental principles of histology, of embryogenesis and of other matters of by no means inconsiderable interest in general biology.

The relation of the smooth muscle cell to the racemose glands in the nasal mucosa differs somewhat from that which obtains in the skin since, according to Koelliker,[84] in the con-

[81]Normale und Pathologische Anatomie der Nasenhoehle, I, 1893.

[82]Beitraege zur Anatomie des Schwellkoerpers der Nasenschleimhaut—Archiv fuer mikrokopische Anatomie, XXXIV, p. 197, 1889.

[83]Heymann's Handbuch der Laryngologie und Rhinologie, III., erste Haelfte.

[84]Gewebelehre, I., p. 91, 1889.

nective tissue network around the skin glands there is mingled with it elastic fibres, fat cells and often muscle cells. It is true that muscle cells can often be made out in connection with the racemose glands of the mucosa, but in health with regularity only around the ducts of the glands and here they are seen even closer up under the surface epithelium at their mouths, the only situation immediately under the surface epithelium, so far as I have been able to observe, where the muscle cell can be demonstrated in the nasal mucosa. We usually have the mouth of the duct at the bottom of a deep sulcus in the mucosa, so that the muscle cell as a matter of fact does not even here usually approach very closely to the surface. Further, it may be noticed in this connection that frequently blood-vessels of considerable calibre (fifty microns estimated in one observation in my notes), have no encircling muscle coat when they lie close under the surface epithelium. It will be remembered that I described as normally existing close under the epithelium a special horizontal layer of elastic fibres, and we may readily believe that these are more serviceable in obtaining a smooth surface on contraction than muscle fibres would be in that situation, while deeper in the stroma a musculature is required to supplement the obliquely arranged elastic fibres. For efficiency in respiration and in olfaction a smooth nasal surface would be very desirable, but for proper drainage it is imperative. I shall not attempt a complete description of the musculature of the blood-vessels further than to say the deep veins and sinuses and deep vessels of even very small calibre are supplied with well developed muscle cells in their coats, both longitudinal and transverse; in the superficial vessels, as I have just intimated, the character of the coats is that of large and small capillaries.

One cannot have constantly before one's eyes the difficulty of distinguishing the indifferent cell from the differentiated muscle cell in the connective tissue without the growth of a conviction that out of the former may arise the latter in answer to some functional demand, increasing in importance as one approaches the vascular layers. In another respect it may quite as rationally, even from my observations, be argued that

the muscle cells have split off primarily from those of the vessel coats, by mitosis, in the development of the bulk of the erectile bodies which takes place at puberty. This might easily occur and the muscle cells might be isolated at considerable distances by the proliferation and interposition of the different connective tissue cells of the loose stroma. In the calf, and in the human infant before birth, especially in the latter, where the embryonic type of cell prevails, the isolation of the muscle cell, if I am to trust my own notes, is even more conclusively to be observed than in the human adult. To be sure there is no reason why the genesis of the stroma muscle cells may not be ascribed to both sources—i. e., to their original embryonic differentiation in the tissue and to their derivation under certain conditions from the musculature of the blood-vessels. The question of the mutation of the indifferent adult connective tissue cell into a muscle cell is quite another matter, and will find little support from some observers. Nevertheless, while I admit that my observations are susceptible of an orthodox interpretation in this instance I am disposed to exclude the possibility of such a metamorphosis, especially in the disturbances of chronic inflammation. In other articles[85] I have dwelt sufficiently upon the evidence of a metamorphosis of epithelial cells into cells exhibiting the fibrils of connective tissue under the stimuli of chronic inflammation. Here, as in the tonsils, chronic inflammation certainly induces an appearance of metaplasia, which has forced itself on my attention. The branching connective tissue cell is much more closely related embryologically to the muscle cell than to the epithelial cell, and how much resistance this view of their functional and morphological mutability may receive I do not know.

This brings me to the fate of the smooth muscle cell in plainly pathological states of the nasal mucosa. I have explained at great length the difficulty one must experience in saying where normal nasal structure in the adult becomes abnormal. A very wide neutral territory must be pictured as existing between the two. With the elastic tissue we have

[85]The Laryngoscope, July, 1909, *et alidem.*

seen that when we emerge out of that neutral debatable strip into the clearly pathological domains of oedematous infiltration and of atrophic change, we have found a very marked diminution not only in the efficient coaptation of the elastic granules and the configuration of the fibres, which occurs early in the course of hyperplastic change, but we have found a marked diminution in the quantity of elastin, organized and disorganized, everywhere in the tissue. Clinically, in the viscid mucus which clogs the gland acini in atrophic rhinitis, we recognize an increased demand for the function of an actively contracting cell around the acinus. Comparing sections differently stained of the same tissue in atrophic rhinitis we at once note in the penultimate stage that the muscle cells have offered a markedly greater resistance to degeneration and absorption than has the elastic fibre. I have pointed out that in the normal tissue the smooth muscle cell, or something resembling it, is seen around the mouth of the racemose gland ducts, but not around the acinus. In the primary changes in the glands in the course of chronic hypertrophy the muscle cell is noted also wrapped around the acini in such a way as to suggest that it is there in answer to a functional demand for the expulsion of a thickened mucus, or possibly to take the place of the elastic tissue which has ceased in the stroma to transmit its pressure to the glandular structures. Passing on to fairly well advanced atrophic conditions, stages in which there has been marked destruction of the venous sinuses and the glands by the encroachment of low grade fibrous tissue and the obliteration of these structures, we note that the muscle cell in the walls of the arterioles and venous sinuses most affected is assuming the irregular nucleus form and branching cell body of the indifferent connective tissue cell. At least from my point of view this is what seems to take place, though I cannot deny the validity of the description which narrates the history of the process in another fashion, viz., that this indifferent connective tissue cell replaces the muscle cell.

A careful study of the cell forms in the stroma of these moderately advanced cases of atrophy in comparison with hypertrophy will reveal comparatively very many more cells,

entirely isolated from the neighborhood of any blood-vessel, which have assumed or preserved the morphology of the smooth muscle cell than in normal or hypertrophic mucosae. It is true, a perfect picture of the muscle cell form is not obtained in the technique I have used, and the criticism is unavoidable that pressure has moulded indifferent connective tissue cells into the rounded ribbon or spindle-like form of the muscle cell with a cylindrical nucleus; but I am sure if they were wound around a blood-vessel there would be no hesitation on the part of any histologist in classifying them as smooth muscle cells. In the later or last stage of atrophy of the mucosa all muscle cell forms have disappeared, the stroma has been reduced almost to the condition of granulation tissue made up of round cells in a feeble mesh of star-shaped branching cells with long processes, resembling the embryonic type of cell and numbers of single walled capillaries.

The structural changes wrought in the mucosa are evidenced in the vitiation of the secretions. The absorption of bone and cartilage is responsible for a change in the external configuration of the nose when the disease has begun in early childhood, before the facial bones are fully developed. Whether it has begun in childhood or at puberty the roominess of the nasal chambers is dependent upon atrophy of the scroll bones and of the mucosa covering them.

How to account for the disappearance of the crusts and the secretions in those cases in which it occurs after middle life remains a subject for histological research. A restoration of the glands and blood vessels anatomically seems biologically improbable, yet there is a return to a secretion on the surface of such a nature that its evaporation is not rapid enough to prevent it from carrying away with it those non-volatile ingredients which formerly were left behind as crusts of dried secretion.

The atrophy of age is of the same structural nature, so far as the glands and blood vessels and the periosteum are concerned, and it seems probable that in those cases which recover from their terrible affliction of foul-smelling nasal crusts, the senile stage of the mucosa is reached and its condi-

RESULTS IN A SERIES OF CASES OF TONSILLECTOMY AT THE MASSACHUSETTS GENERAL HOSPITAL THREE TO FOUR YEARS AFTER OPERATION.

BY J. PAYSON CLARK, M.D.

The year beginning July 1st, 1908, was chosen for study, because sufficient time has elapsed since then to make the reports valuable as end results. The reasons for not selecting an earlier year were, in the first place, that this was about the time when we began to do tonsillectomy at the hospital as a practically routine procedure in all cases where a removal of the tonsils appeared to be indicated; and, in the second place, because the longer the time since the operation the smaller the number who were likely to respond to a request to report. The reasons for this latter statement are obvious and need not be mentioned. Postal cards stating, in substance, that Dr. Clark was interested to know their present condition and would like to have them report on one of certain given dates at the Throat Clinic of the Hospital were sent to all the cases. Those selected were under the heading "Adenoids and Tonsils" in our card index. Of course, many of these cases had only an adenoid operation, and such cases were not asked to report. As a result of selecting only the cases of tonsil operation which were listed under this heading, it is obvious that adults were practically excluded from the list. It was felt that as the tonsil is physiologically more active in children, a study of end results would be more valuable in them, and that the results of tonsillectomy in adults could more profitably be considered in a separate paper.

I began sending out these notices in July, 1912, sending out a few each week, so that I should not be overwhelmed by too

transudation to the surface of lipo-proteids, derived from tissue waste and perverted gland function, which form a stinking, viscid secretion and crusts in which bacteria plentifully multiply.

10. Finally, this destructive metabolism ceases with the advent of old age. The foul secretions and crusts disappear and the surface is moistened by a serous exudate which dilutes the remaining gland secretion sufficiently to secure its drainage. Thus nature brings about a cure difficult or impossible for man to accomplish.

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I began sending out these notices in July, 1912, sending out a few each week, so that I should not be overwhelmed by too

large a number reporting at any one time. I wanted to see all the cases myself, but this was not possible, owing to my being unable to go to the hospital at several different times. The cases which I did not see personally (53 in all) were examined by one of my colleagues and recorded for me. Nine hundred and ninety-two postals were mailed, and to these 143 patients responded by reporting in person at the clinic. This is only 14 2-5 per cent of the number asked to report. This percentage is a little better than a few years ago, when I wrote on "Results in Adenoid and Tonsil Operations" (Boston Medical and Surgical Journal, September 24th, 1908). At that time I sent out 595 cards and, to quote from that paper, "The responses were exceedingly disappointing as to number, amounting in all to 73, or a little over 12 per cent of the cases to whom notices were sent. Between three and four years having elapsed since the operation, many of the patients have doubtless moved, so that the cards did not reach them, and others live at too great a distance to be able to come in easily." In talking to other investigators of all kinds of medical and surgical cases I find that my experience in this particular is a common one. I believe, however, that it is fair to take the cases which did report as a pretty good criterion of the results in all the cases. I received a dozen or more letters in response to the card, stating that the patient could not come to the hospital for one reason or another. These cases are, of course, not included in my statistics. Those who reported were subjected to the following examination:

1. Patient's name, sex, age (at present time), number?

2. Date of operation?

3. Any hemorrhage after operation requiring treatment? If so, what treatment?

4. Was adenoid removed at same time or not?

5. For what reason were the tonsils removed? (On account of size, frequent tonsillitis, enlarged cervical glands, rheumatism or other evidence of septic absorption or any other reason?)

6. Was the trouble for which the tonsils were removed relieved?

7. Was there a marked improvement in general condition after operation?

8. Has the patient had any illness since operation? If so, what and when? Has the patient had any sore throat?

9. Present state of health?

10. Appearance of throat. (State whether any tonsillar tissue appears to remain, also as to symmetry of soft palate and pillars, presence or absence of tonsillar fossae, etc.)

11. Any difference in voice or speech since operation?

12. Are there any enlarged cervical glands now?

A review and analysis of the answers to the questions and the findings at the examination follows: The ages of the patients ranged from 5 to 36 years, distributed thus: 5-10 years, 45; 10-15 years, 76; 15-20 years, 17; over 20 years, 4. As these are the ages at the present time, it is obvious that the ages averaged four years less at the time of the operation. Of the patients over 20, one is now 23, one is 26, one 27 and one 36 years old. There were 76 males and 67 females. All who reported had adenoids removed at the same time. It is hardly necessary to say that the adenoids were of varying relative importance in the different cases, sometimes being so small as to seem to be hardly worth considering, and in other cases being the chief reason for operation.

Only one case had bleeding after the operation which was worthy of note. In this case the bleeding was so free that the patient was kept in the hospital over night, but no operative treatment was required to stop the bleeding. I might say here that up to last Fall it was our custom at the Massachusetts General Hospital to allow children on whom tonsillectomy had been done to go home in the afternoon, the operation having been done in the morning. It was felt that if there was no evidence of bleeding by that time it was fairly safe to assume that there would not be any. Owing to the small number of beds at our disposal, we were forced to take this risk or else turn away the majority of our tonsil cases in children. An exception, however, was made in the event of

children living at such a distance from the hospital that they could not be easily reached in case of secondary hemorrhage. These statistics tend to show that the risk which we took was a small one. Since last Fall we have had more beds put at our disposal and now keep all our tonsillectomy cases in the ward for one night. This routine is certainly more conducive to one's peace of mind and much fairer to the children. For, although I feel that where tonsillectomy is competently and carefully done, hemorrhage coming on after the patient has left the operating room and requiring treatment is really a rare occurrence, yet when it does occur it is often so alarming and prompt attention is so important that to keep such patients under observation for twenty-four hours is not being over cautious. The following table shows for what reason the tonsils were removed and whether the conditions were relieved or not:

——————Sore Throat——————			
Unknown.	Relieved.	Not relieved.	Cured.
15	10	4	29
——Adenitis——		——Tonsilitis——	
Size.	Cured.	Not cured.	Cured.	Not cured.
96	15	17	32	1

Chorea.	Rheumatism.	Speech.	Circumstons.	Abscess.
Improved.	Cured.	Cured.	Cured.	
1	2	1	2	

Of the fifteen cases in which the reason for removal is not known several were no doubt operated on because they were under an anaesthetic for an adenoid operation and it was thought just as well to get the tonsils out, too, without considering whether there were good reasons for their removal or not. We endeavor to guard against unnecessary removal of the tonsils by requiring that the man who operates shall see that the reasons for removing the tonsils are distinctly stated on the history. There was a marked improvement in the general condition after the operation in 89 cases, a moderate improvement in 29 cases and no improvement in 24 cases. Since the operation one patient has had nasal diphtheria, four measles, two whooping cough, one neuritis, one "congestion

of the lungs,'' one chorea, one bronchitis, one pleurisy, five abscess of the ear (two of these requiring a mastoid operation), three pneumonia and twenty-two sore throat. The present state of health is good or excellent in 118 cases and fair in 22 cases. There has been no change in the voice or speech in 116 cases. In 22 cases the voice or speech has been improved. In two the voice or speech is said to be not so good. Enlarged cervical glands were absent in 98 cases and present in 43. In two cases there is no record on this point. Tonsil tissue was absent in 82 cases. Some tonsil tissue was still present on both sides in 31 cases and on one side only in 28. Examination was unsatisfactory in the case of an unruly child. Certain cases where it was doubtful whether or not the appearance in the tonsillar fossae was due to tonsillar tissue were listed as showing it. In some of these cases it is fair to assume that the lymphoid tissue which was apparently present in the fossa was due to the hypertrophy of some focus of lymphoid tissue which lay outside the tonsil capsule. The soft palate was symmetrical in 120 cases and asymmetrical in 18. In five cases no note was made on this point. The soft palate was considered asymmetrical when the uvula was at all to one side of the median line or when one arch of the velum was flatter than the other. The pillars of the fauces were normal in 96 cases and not normal in 41. There was no note on this point in 6 cases. The pillars were recorded as not normal when one or more of them were absent or when the anterior and posterior pillars on the same side were fused or when scar tissue showed that one or more of them had been injured at the operation. The tonsil fossae were present on both sides in 95 cases, shallow in 16 and one or both were absent in 26 cases. There was no record of their condition in five cases. Carious teeth were noted as present in 21 cases. Three cases which had enuresis before the operation were not relieved of this trouble. In four cases the uvula was partly or wholly cut off.

Now let us analyze some of these results and see if we can obtain any interesting or valuable facts from them. In the fifteen cases in which the reason for removing the tonsils

does not appear they may have been removed, because they were visible and the operator was of the opinion that as the patient was under ether for an adenoid operation the tonsils were better out, too. Or it may have been that there was some excellent reason for their removal which the operator failed to record. I have already stated how we try to guard against any unnecessary removal of the tonsils. "Sore throat" was given as a reason for removal of the tonsils in cases in which it was not clear as to whether there had been definite tonsillitis, yet on inspection it appeared probable the tonsils were the seat of the trouble. These numbered 43 in all. Twenty-nine of these cases have had no sore throat since the operation, 10 have had it less frequently or less severely and 4 say they have been no better. Of the 10 cases which were relieved, one, a woman of 27 years of age, had suffered from frequent tonsillitis before, but since the operation says she has slight sore throat at times. An examination of this case showed in the fossae a very small amount of tissue, with a suggestion of lymphoid characteristics. The patient had not reported at the time of these attacks of sore throat, and it seems fair to assume that they might have been simple pharyngitis. Of the remainder, five showed the presence of more or less tonsil tissue in one or both fossae. But in all these cases the sore throat was so slight and so infrequent as not to require medical attendance or keep the patients from their regular employment. Four who have occasional slight sore throat showed no tonsil tissue. The sore throat in these cases was probably a pharyngitis. Of the four cases in which the sore throat was said to be no better, two showed absolutely no remains of either tonsil, and one of these had a marked chronic pharyngitis. One of the other two showed a small remnant of tonsil on the right, but it seemed very doubtful if this could be the origin of the sore throats which the patient said he had, but for which he had never applied for treatment. In the remaining case there was some doubtful lymphoid tissue in the right fossa. Of the 32 cases in which cervical adenitis was mentioned among the reasons for tonsillectomy, 15 were cured—i. e., they show no enlarged cer-

vical glands now. A further consideration of the 17 cases of adenitis which are put down as not cured discloses some interesting facts. But it will perhaps be more satisfactory to consider these cases later, in connection with those in which the presence or absence of cervical glands was not mentioned at the time of the operation but in which cervical glands were felt at this examination.

Of the thirty-three cases in which frequent tonsillitis was given as a reason for tonsillectomy only one was not cured and in this case it was found that the tonsils had been only partially removed. A case which came on account of deafness was cured, but the simultaneous removal of the adenoids was no doubt a larger factor in this result. One case of chorea is much better, but not completely cured. Two cases of rheumatism are cured. It is only fair to say that in neither of these cases was the history of rheumatism very clear. Three cases of circumtonsillar abscess have been cured; one now being 36 years old, one 26 and one 17. Only one patient under 21 years old had circumtonsillar abscess. In eighty-nine cases there was marked improvement in the general condition after the operation. As the adenoids were removed at the same time, a part of the improvement must be attributed to this cause in many of these cases. In twenty-nine other cases there was some improvement, but not marked. In twenty-four cases there was no improvement in the general condition. In fourteen of this group the health was good before the operation and the reason for operation was a purely local one. One case of quinsy is cured of that affection and has no tonsil tissue remaining. She now complains of pain going to different parts of the body, on which no diagnosis has been made. One case has no tonsils, but a marked nasal obstruction due to a deviated septum, which may account for lack of general improvement. One case shows no tonsils but very bad teeth, which are undoubtedly the cause of poor general condition. One patient, whose tonsils have been completely removed, is pale and nervous from some other cause, probably improper diet and carious teeth. One case of retarded mental development was not improved by the operation. One patient,

described as "nervous and fidgety" by her mother before the operation, shows no improvement in this respect. Her tonsils have been completely removed. The neurologist describes the condition as an "involuntary twitching of the shoulders, characteristically repeated." Diagnosis, "Habit spasm." Four of these cases still show presence of some tonsil tissue on one or both sides. In one of the four it seems more than probable that the present anemic condition and loss of appetite are due to bad teeth and chronic rhinitis rather than to the small amount of the lower lobe of each tonsil which appears to be present. In the other three cases it is fair to assume, in the absence of other obvious cause, that the lack of general improvement is due to the incomplete operation.

Among the cases which reported there have been the following illnesses: Nasal diphtheria, one; measles, four; whooping cough, two; pneumonia, three; "congestion of the lungs," one; pleurisy, one; bronchitis, one; neuritis (?) one; chorea, one; abscess of the ear, five cases (two of which had a mastoid operation). Several had more than one illness, so that the actual number of individuals in the above list is only sixteen. It strikes me as a remarkably good record that of one hundred and forty-three children only sixteen have had a definite illness in four years. The present state of health is stated as good or excellent in one hundred and eighteen cases, fair or poor in twenty-two. Ten of these latter showed definite remains of tonsil in one or both fossae, while three were doubtful. The presence of some tonsil tissue may account for the poor health in some of these thirteen cases, while in the nine others in which there is no tonsil remaining one has chorea; three, bad teeth; two, nasal obstruction, and one has just recovered from an illness. There was no change in the voice or speech in one hundred and sixteen cases, including the four in which the uvula had been partially or wholly cut off. This accident has had no ill effect save a cosmetic one in any case. In twenty-two cases the voice or speech was improved, i. e., it is clearer or more distinct or of better quality. In two, the voice or speech is said not to be so good. One boy talks somewhat through his nose, which he is said not to have done before the

operation. His soft palate, uvula and faucial pillars are normal, but he has a nasal obstruction, which probably accounts for the nasal voice. The other is said to talk at times through the nose, but there is no apparent reason for this, as his throat is perfectly normal.

Forty-three cases showed one or more enlarged cervical glands. In not one of these cases were the glands causing any apparent symptoms or inconvenience of any sort. They were discovered on palpation of the neck. In seventeen cases a few small glands were present on both sides. In thirteen of these tonsil tissue was left on both sides. In three of the thirteen there were also decayed teeth. In five cases there were decayed teeth and no tonsil. In five others there was no tonsil tissue and no decayed teeth. In four cases there were several glands, and in eleven cases only one palpable gland under the *left* angle of the jaw. Eight of these fifteen cases showed the presence of a remnant of the tonsil on the left, and in one there was also a decayed tooth. In four there were decayed teeth and no tonsil, and in two there were no tonsil and no decayed teeth.

In three cases there were several and in five cases one gland on the right. In three of these eight cases there was some tonsil remaining on the right and in three no tonsil but decayed teeth. In these cases it is interesting to note that in most of the cases where there were glands present there was still some tonsil on the same side. In five of the cases in which glands were felt it was doubtful whether or not tonsil tissue remained. On the other hand in twenty-six cases in which there were remains of the tonsil on one or both sides there were no enlarged cervical glands. In four of the cases in which there were decayed teeth there were no enlarged glands.

Case. S. B. F., female, age 8½ years, was seen in the neurological department in March, 1909, where the following note was made: "Patient has general chorea, twitching of both arms and both legs so that she cannot walk, her mother says, or feed herself. Heart negative. Advise operation for tonsils." She also gave the history of frequent tonsillitis. Her

tonsils were removed on April 1, 1909. When she came in to report it was stated that she was much improved in choreaic symptoms, but not cured. On examination no tonsil tissue could be seen. She had one or two teeth with cavities and one pea-sized gland under the left angle of the jaw. She was sent to the dentist.

Summary.

The patients with a few exceptions were under 15 years old at the time of operation.

Hemorrhage after tonsillectomy, calling for special treatment, is of rare occurrence.

The condition for which the tonsils were removed was relieved in very nearly every case, even in those in which there is still some tonsil tissue.

An improvement in the general health is to be expected after tonsillectomy done for cause.

Children who have had tonsillectomy certainly show no increased tendency to illness and are probably less susceptible than before the operation.

Present health is excellent in the great majority of cases.

What is apparently tonsil tissue is found much more often than one would expect after supposed tonsillectomy.

The soft palate was symmetrical and the faucial pillars and tonsil fossae normal in the great majority of the cases.

The accidental cutting off of the uvula in four cases caused no bad symptoms. Most of the cases of sore throat and tonsillitis were relieved by the operation.

In many of the cases in which there appeared to be remains of tonsil tissue the patients are in perfect health and in others in which there were symptoms these were undoubtedly due in many cases to other causes than the tonsil remnants.

The ordinary voice or speech may be said to be practically unaffected by tonsillectomy. No investigation was made as to the singing voice.

In many of the cases in which enlarged cervical glands could be felt there was tonsil tissue present on the same side. On the other hand, in nearly half the cases in which tonsil tissue is present there are no enlarged glands.

Carious teeth were apparently a direct cause of some cases of cervical adenitis.

THE FAUCIAL TONSILS AS A FOCUS FOR SYSTEMIC INFECTION.

BY GEO. E. SHAMBAUGH, M.D.

The close relation between acute rheumatic fever and sore throat has long been recognized. Formerly it was often assumed that the sore throat occurring in connection with acute rheumatism was but a local manifestation in the pharynx of the general systemic condition. It is now generally believed, however, that the sore throat in these cases represents the focus of entrance for the systemic infection. Furthermore it is now quite generally recognized that sore throat, for the most part acute tonsillitis, is very often the immediate cause for other systemic infections such as acute endocarditis and acute nephritis. It has not been so generally appreciated that the faucial tonsils are very frequently the focus for chronic systemic infections, such as chronic arthritis, commonly known as arthritis deformans, chronic neuritis, cardio-vascular degeneration and chronic nephritis.

In the Archives of Internal Medicine, April, 1912, Dr. Frank Billings, of Chicago, discusses the subject of "Chronic Focal Infections and Their Etiologic Relations to Arthritis and Nephritis." This work was done on patients in the Presbyterian Hospital of our city, and it has been my privilege to examine the throats of many of them and to enucleate the faucial tonsils in certain of the cases where these structures were suspected of harboring foci for the systemic infection. The observations on these cases and on a large number of other cases of focal systemic infection referred to me during the past few years have impressed on me certain facts; first, as regards the relation of the faucial tonsils to systemic infection; second, as regards the conditions found in the tonsils in cases where these structures were clearly shown to harbor foci for systemic infection, and third, as regards the proper treatment for tonsils suspected of containing foci producing systemic infection. In this paper

I wish to discuss briefly some of the conclusions which I have reached regarding these questions.

In the first place I believe that the general practitioner, as well as the specialist, has not, as a rule, fully appreciated the importance of the relation existing between infections, acute as well as latent, of the faucial tonsils and certain systemic conditions. I believe that in general the importance of this relation has been more fully appreciated by the leading internists than it has by the specialist. This is, perhaps, to be expected because the consulting internist is the one to whom such cases are referred. The internist is, moreover, familiar with systemic conditions caused by other foci of infection, such as abscess of the gums, chronic appendicitis, infection of the genital tract, cholecystitis, etc. The internist is, therefore, prepared to recognize in a structure like the faucial tonsil, which is so frequently the seat of violent acute streptococcus infection, not only the source for acute systemic infection, such as acute rheumatic fever, acute endocarditis and nephritis, but also the seat of persistent latent chronic foci of infection capable of causing chronic arthritis, chronic neuritis, cardio-vascular degenerations, nephritis, etc. The attention of the specialist, on the other hand, is naturally centred more on the purely local conditions. He cannot be expected to have that fuller appreciation of the systemic conditions which experience brings to the internist. It is so easy for the specialist to conclude that because he has not in his experience recognized systemic conditions resulting from tonsil infection that therefore these systemic conditions do not result from disease of the tonsils. No greater mistake in reasoning than this can be made. It would be just as logical for an otologist to assert that because he has never seen a brain abscess develop in any of his cases of purulent otitis media that, therefore, purulent otitis media is not the chief cause of brain abscess. Dr. Billings states: "There can be no other reason for the prevalence of rheumatism found in childhood than the frequency of local infections in the throat and nose. Quite as frequently children have endocarditis without other symptoms of rheumatic infection, which has its source in the throat. . . . 1

think any one who has noted the difference in the physical and mental makeup of a child before and after the removal of large amounts of lymphoid tissue in the throat must be convinced that the change is due not only to a better air space, but also probably to the removal of a constant, even though it may be slight, systemic infection."

As regards the appearance of the faucial tonsil which is a focus for systemic infection, it is quite clear that the size of the tonsil is no index of the menace this structure may be to the individual. A large tonsil is often chronically diseased or the seat of repeated violent acute infections and, therefore, a constant menace as a focus for systemic infection. On the other hand one sees a great many very large tonsils where the whole appearance is one of simple hypertrophy and where there is no history of attacks of acute tonsillitis. The danger from tonsils of this type is as a rule very slight when compared with the danger from a small tonsil which is subject to repeated acute infections. At the same time the children with these enormous, apparently harmless, faucial tonsils often do appear ill-nourished and generally below par. Very frequently, too, in such cases the removal of the tonsils results in such an immediate and astonishing improvement in general health that it can hardly be accounted for except on the assumption that these enlarged, harmless appearing tonsils often contain foci for a mild systemic infection. The enlarged tonsil in the adult, which I have come to regard with suspicion as the possible focus for chronic systemic infection, appears often more or less oedematous and congested. The surface is more irregular, caused at times by deep ragged fissures, at other times by swelling of the parenchyma between the meshes of hypertrophic connective tissue stroma. In many of the tonsils there are dilated crypts containing the characteristic foul smelling cheesy deposits which in many cases from time to time produce circumscribed acute infection, as shown by the local congestion and soreness. At such times the patients experience more or less general malaise and dullness. Not infrequently one can, by pressing on the base of a tonsil express from the depth a creamy fluid exudate which is largely pus, often filled

with virulent streptococci. These enlarged tonsils in the adult are frequently quite fibrous and hard, but in some the swelling is largely of the parenchyma and the tonsils remain soft and compressible.

Another suspicious type of tonsil frequently met with in adults is the very small fibrous tonsil. This condition is found in individuals who, for many years, have been subject to a great many attacks of acute tonsillitis. I have several times removed tonsils which were so shrunken as to represent scarcely more than a thin band of fibrous scar tissue, from which the macroscopic evidence of parenchyma had been largely eliminated and where the structure was so firmly adherent to the underlying tissue that it could not be pulled out of its bed, as is done before dissecting the tonsil free from the pillars. The patient is often not aware that the recurring attacks of sore throat are in reality recurring attacks of acute infection in the faucial tonsils. It is only by seeing the patient during an attack of sore throat that the real character of the trouble can be diagnosed. The amount of local discomfort, as well as the general symptoms, fever, headache, etc., are, as a rule, not very severe as compared with attacks of acute tonsil infection occurring in an enlarged faucial tonsil. I have seen a number of cases of chronic arthritis and chronic neuritis occurring in patients who had for years suffered from this type of tonsil trouble, where each fresh attack of tonsillitis was followed in a few days by an acute exascerbation of the chronic systemic infection and where the enucleation of the small insignificant looking fibrous tonsils resulted in a complete cessation of attacks of acute sore throat as well as a disappearance of the chronic systemic infection and a remarkable improvement in the patient's general health.

Every one is familiar with the small tonsil embedded under a fold from the anterior pillar and the tonsil with a deep horizontal fissure separating the upper from the middle thirds, and with the fact that such tonsils are unusually susceptible to acute infections which, therefore, especially predispose them to the development of latent foci capable of causing systemic infection.

There is another type of faucial tonsil that I have learned to regard as a frequent source for systemic infection. This is the tonsil stub remaining after partial removal or where the tonsil has been subjected to ignipuncture or surface cauterization. In these cases the surface scar seems often to predispose to the retention of infection in the pockets of the remaining stub, causing repeated attacks of acute tonsillitis, as well as systemic infection.

Occasionally one observes near the surface of the tonsil a sealed pocket filled with yellow pus. The tissue immediately surrounding such a pocket of pus often shows from time to time an acute reaction with swelling and congestion. On the other hand such pockets are often observed when there is no evidence of an acute condition in the tonsils which would suggest tonsil infection. Pockets of this sort in the faucial tonsils frequently constitute the foci for chronic systemic infection. The size of the pocket of pus is not an important factor. Small foci seem to be as dangerous as a larger abscess. It is often overlooked that such foci may develop in the depth of a tonsil. As a matter of fact one discovers such foci much more frequently near the bottom of the tonsil than near the surface. It would seem that the foci developing near the surface of the tonsil find an opening and heal more readily than when they develop near the bottom of the crypts. The presence of latent foci can sometimes be suspected from the appearance of the tonsil as described above. On the other hand I have often been surprised after removal of a tonsil suspected of causing systemic infection to discover near the base a pocket of pus, when neither the surface appearance of the tonsil nor the history suggested tonsil infection. Such experiences have taught me not to be too dogmatic in asserting that a tonsil may not be the focus for systemic infection simply because there is nothing suspicious in its appearance. In this connection I wish to call attention to a condition which may occasionally follow what seems to be a complete enucleation of the tonsil. This is the formation, in the process of healing and contraction, of a small fistula which later may become the seat of an acute purulent infection with a circumscribed

abscess formation in the bottom of the fistula. This condition I have seen in three cases. In two of the cases the abscess was located about the middle of the tonsil region and the acute reaction occurred more than a year after the tonsil enucleation. In the third case the fistula was observed three months after the tonsil operation. There had been no acute symptoms and the fistula led to a small cyst, the size of a pea, filled with pus, containing staphylococcus and located in the region of the supra-tonsillar fossa.

Chronic foci of infection in the faucial tonsil, just as such foci occurring elsewhere, represent a condition which has had its origin in some acute infection. In most of the cases the patient gives a history of such acute infections, consisting in attacks of acute tonsillitis. In many of the cases with chronic foci in the tonsils there may not have been an acute attack of tonsillitis for a number of years. In all cases of chronic systemic focal infection, where the competent internist is unable to discover evidence of foci elsewhere, the faucial tonsil should be suspected. Usually when the tonsils are at fault there will be a history of attacks of sore throat, tonsillitis, and a careful examination of the tonsils will disclose evidences of chronic infection.

As regards the proper treatment of faucial tonsils suspected of harboring foci giving rise to systemic infection, this should be the same as the treatment of such foci of infection occurring elsewhere in the body. The treatment consists in the thorough removal of the suspected foci. In the case of the faucial tonsils, I know of but one way by which this result can be assured. This consists in the thorough enucleation of the faucial tonsils. Whatever theoretical arguments may be urged against tonsillectomy as a routine procedure when operating on enlarged faucial tonsils in children, such arguments should not prevent us from the thorough enucleation of faucial tonsils, whether they be large or small, when these structures are suspected of harboring foci causing systemic infection. Only in those cases, especially in adults, where the danger of a serious bleeding is increased because of a high blood pressure or a slow coagulation time, should the enucleation of the tonsils

give place for less radical measures. Here we must be content with efforts to cleanse the tonsil pockets and possibly also with cautiously slitting them open. Surface cauterization or ignipuncture should never be resorted to, as these often tend to increase the trouble.

The enucleation of the faucial tonsils in children requires a general anaesthesia. In adults I prefer, as a rule, to do the operation under local anaesthesia for the following reasons: The operation can be done with the patient in the upright position; the annoyance from bleeding is very much less; the operation can be done with less traumatism to the surrounding structures, and the discomfort from soreness is less than where general anaesthesia is employed. All cases are operated at the hospital. The patient remains at the hospital over night, or for several days if they wish. I begin usually about 2 P. M. When local anaesthesia is to be used, the patient goes without lunch in order to decrease the discomfort from gagging while working in the throat. A hypodermic containing 1-6 gr. of morphine with atropine is given just before the patient is brought to the operating room. I use a 5 per cent solution of cocaine in 1-1000 adrenalin, rubbed over the attachment of the tonsils. A ½ per cent solution of novocain, to which a few drops of adrenalin are added, is freely injected under the mucous membrane along the margin of attachment of the tonsil on both the anterior and posterior pillars. A deep injection of this solution is made in the lymphoid tissue at the lower part of the tonsil, and another is made in the base of the tonsil directly through the anterior surface of the soft palate. This part of the work is done deliberately, and both tonsils are prepared for operation before an incision is made. I have used this method for over three years, and have never seen any symptoms of poisoning such as an injection of even a small amount of cocaine usually produces. Both tonsils are dissected free from the pillars before either is removed with the snare. In most cases the only part of the operation that is at all painful is when the snare is being used. For this reason it is important that the difficult part of the operation, the freeing of the tonsils from the pillars, should be done before

either tonsil is enucleated. After a patient has once been hurt by the removal of one tonsil he may be too nervous to allow the careful freeing of the second tonsil from the pillars. The separation of the tonsils from the pillars is accomplished with the least discomfort to the patient by using a straight scalpel, very sharp and with a rounded end. I find that in separating the left tonsil from the posterior pillar and the right from the anterior pillar, it is an advantage to hold the scalpel in the left hand while the tonsil forceps are held in the right hand. The patient holds the tongue depresser. The snare to be used when removing the tonsils under local anaesthesia should be one that can be closed instantly if desired. The slow closure of the snare is often too painful when local anaethesia is employed.

Discussion on Drs. Shambaugh's and Clark's Papers.

Dr. G. Hudson Makuen, of Philadelphia: We have heard two very interesting papers on the tonsils, one dealing particularly with infection and the other dealing with operations. There are two reasons for operating upon the tonsils, the first one being for the removal of sources of infection, and the second one for the conservation and improvement of the functions of important organs of speech and voice.

It is interesting to note in this connection that in the London Lancet, of February 15, 1913, Dr. Harold Whale reports a study of two hundred and twenty (220) unselected cases with reference to the effect of the tonsil operation upon the voice, and he states that of all those in which the voice troubles were cured nearly one-half presented considerable tonsillar remains, and that the percentage proportion of uncured cases was nearly twice as great after tonsillectomy as after tonsillotomy. From these investigations he concluded that tonsillotomy was the operation of choice for the cure of vocal defects.

This accords with my own experience, and I am of the opinion that tonsillectomy should be the operation of choice only for the removal of sources of infection and that so far as the voice is concerned better results are obtained by leaving the capsules and possibly some portions of the tonsil in the fauces to keep the pillars of the palate apart and direct their action in the control of the vocal cords. I have yet to see perfectly functionating palatal pillars after a complete removal of the tonsils.

All these discussions should be not on "tonsillectomy vs. tonsillotomy," but on tonsillectomy vs. all other operative measures. Tonsillotomy, or the mere cutting off of a portion of the tonsil, including, as it often does, the edges of the palatal pillars, is to be condemned together with what Dr. Shambaugh has called surface cauterization, because they both tend to seal the crypts and form infective pockets.

My own practice, when a tonsillectomy or a complete removal of the tonsil is not absolutely indicated, is to insert a specially devised knife having the blade at nearly right angles to the shaft deeply into the tonsillar crypts and slit them freely open down to the very base, after which a thorough searing of the crypts and the cut edges by means of the cautery knife follows in order to prevent adhesions between the edges of the wound. Each crypt may thus be opened up in turn, and, if necessary, at different sittings. These operative measures result in a marked shrinkage of the tonsil without forming adhesions of any kind and without interfering at all with the action of the palatal muscles.

In my own experience tonsil operations are done quite as frequently for the relief of speech and voice troubles as for the removal of focus infections, and I find that the twofold purpose for any tonsil operation is often accomplished by such conservative measures. Should a focus of infection develop later it may be found and opened up at any time. These conservative measures are better adapted, of course, to adult cases, and in adult cases they may be done in the office under local anesthesia and without subjecting the patient to much, if any, pain. In operating upon children under general anesthesia, I prefer to remove as much as possible of the tonsil without disturbing the capsule, and the only objection to this operation is the fact that it is somewhat difficult to perform.

Dr. William E. Casselberry, of Chicago: I wish to mention the extreme susceptibility of certain individuals to acquire general infection not only from the faucial tonsils as a focus, but from any other of the lymphoid glands of the throat and also from the nasal accessory sinuses. I recall a case of a "rheumatic" subject who, after the tonsils were removed, remained free from rheumatic symptoms for a number of months. He then had acute nasal accessory sinus suppuration with a return of arthritis and other general symptoms, which again promptly disappeared after effective drainage of the group of nasal sinuses involved. I have already called attention when speaking of total tonsillectomy to the large amount of tonsil-like tisue in the throat in addition to the faucial tonsils, so that

even after total tonsillectomy very susceptible persons will have pharyngitis lateralis, affecting the chain of lymphoid glands behind the posterior pillar, or may have an inflammation of the lingual tonsil, either of which can occasion general arthritis and symptoms like those of tonsillitis, but usually milder in degree. This explains the recurrences of sore throat, which seem to the patient so surprising after the tonsils have been well removed.

Dr. Cornelius G. Coakley, of New York: I was very much interested by the papers just read. Dr. Shambaugh's paper was very much in accord with the views of the men in general medicine in New York City to-day—they are looking for the source of infection in the throat when they are unable to find it elsewhere. I find that it is often very difficult to determine whether or not the tonsils are diseased. If one takes an ordinary tongue depressor and examines the tonsils one gets no idea of the pathological process. The only way, in my opinion, is to take a probe, bent at right angles, and force the tonsils from their bed by pressing external to the anterior pillar. From various portions of the tonsils, by means of a little milking, the secretion will come out; then when I take a culture it is surprising how frequently the presence of streptococci is reported. It is also remarkable how often the removal of these tonsils is followed by a sense of general well being and a disappearance or at least a diminution in the symptoms pointing to some chronic infection. I feel with Dr. Shambaugh that the stumps of all partially removed tonsils frequently give trouble. The old-fashioned idea regarding what is the proper operation to pursue has gone, such as the burning or the application of strong solutions of silver nitrate or caustics. It has been my misfortune to see a larger number of secondary operations upon the tonsils this past winter than during the two previous years, and whether this is due to some general sepsis or not I do not know. Seventy (70) per cent of the operations this year have been upon children and young adults who have been operated on once or twice before by tonsillotomy. This is a rather high percentage for secondary operations.

In regard to the method of procedure in children and in adults, Dr. Shambaugh has assured us of the advantages of the use of local anesthesia, and I have felt the same way until some three years ago, since which time I have been using general anesthesia and have gotten little more hemorrhage in adults than I have in children. If one carefully dissects the pillars, then takes a wad of gauze which is folded, rolling it into a sphere somewhat larger than the enucleated tonsil and placing

it in the wound after the operation, hemorrhage will be controlled. It is surprising how large a wad of gauze can be applied and stay there—the pillars of the fauces holding it there. This is left in place and attached by a string. Then one can go ahead and dissect out the other tonsil. Such a pad will stay in place for one or two hours; often even when the patient has vomited the pad will remain in place. It is astonishing how these pads will prevent hemorrhage.

We all appreciate the work done in preparing such statistics as Dr. Clark presented to us and they are valuable. At the clinic we operate at 1:30 o'clock, but the patients do not leave until 4:30 or 5 o'clock, and we meet with but very few cases of hemorrhage.

I believe that Dr. Casselberry hits the nail on the head in what he states regarding sore throats.

I would like to ask whether the cases after tonsillectomy are more susceptible to scarlet fever and other infections. This is a point that has been much discussed. Work is now being done at the Rockefeller Institute, in New York City, in which I am interested, and I am wondering just what the effect of the removal of the tonsils will have in patients exposed to these infections, such as scarlet fever.

Dr. James E. Logan, of Kansas City: Dr. Shambaugh's paper interested me very much. The general physician brings this tonsil question to our attention constantly and is demanding their removal for general systemic disturbances. I believe that many functionating tonsils are removed, and I do not approve of removal when not diseased. The size of the tonsil does not always indicate the amount of disease.

Dr. Clark's paper is of great interest and his statistics are important to remember. It is my belief that tonsils are given more importance with reference to systemic infection than clinical facts bear out. Infections of the upper respiratory tract most frequently come from the presence of adenoids, and this applies to adults as well as children. I often remove adenoids in children and leave tonsils that I believe to be functionating.

The radical adenectomy is just as important, if not more so, than the radical tonsillectomy. The complete removal of tissue in the fossae of Rosenmuller and in and about the Eustachian lips is absolutely important.

Dr. E. Fletcher Ingals, of Chicago: Referring to what Dr. Shambaugh has said regarding tonsillitis, I am satisfied that in many of these cases a very desirable procedure is to pass the cautery point deep into the crypts and move it about so as to cauterize the whole inner surface of the crypt and thus cause

an obliteration of the follicle. I have found this a better treatment than complete removal of the tonsil in many cases. What Dr. Logan has said is not far wrong.

Dr. Henry L. Swain, of New Haven: One should be full of enthusiasm after hearing the remarks of the last two speakers, but we can not arrive at any final conclusions until we have more statistics such as Dr. Clark has presented to us. The question has been raised regarding the effect of the complete removal of the tonsils and adenoids in quite young children, and I believe with Drs. Logan and Ingals that they answer some purpose in the throat, a fact to which laryngologists and men in general work should pay heed. It is a routine practice in some hospitals to take out all adenoids and tonsils whenever patients are sent in for operation. What we should endeavor to learn is through very careful investigations of children who have gone through the radical operation of removal of tonsils and adenoids, under 6 or 8 years of age, if they differ, and how they differ from those who have not been subjected to a tonsillectomy and removal of adenoids. This might be done in large clinics where we can get at much material for such an investigation.

In regard to Dr. Casselberry's remarks that there is something else which causes a sore throat, other causes of infection than tonsils and adenoids, there is much food for thought.

Dr. John O. Roe, of Rochester: No one with any knowledge of or experience in throat work can disagree with Dr. Shambaugh that the faucial tonsils are frequently the seat and focus of systemic infections, and that the only successful treatment for this condition is the radical removal of the entire tonsil. The crypts extend to the bottom of the tonsil; consequently, when the crypts are diseased, the tonsil is diseased all the way through and no other treatment will be effective.

Regarding the question as to the degree of systemic infection from such tonsilar disease, it may be said that this depends upon the condition of the tonsil. While we may consider all enlargements of the tonsil as the result of pathological process, we may have in the one case a simple hyperplasia of the stroma with but little or no infection of the crypts, whereas in other cases the process of the disease may be confined almost entirely to the crypts, with but comparatively little hyperplasia of the stroma. Therefore, the question as to the necessity for a radical operation in any particular case depends upon the diagnosis. In this hypertrophic form, the radical operation may not be necessary, and it might be advisable in such cases as those mentioned by Dr. Makuen to leave a portion of the tonsil, especially in cases of vocalists, so that

there might be less cicatricial contraction following the operation, and consequently less rigidity of the throat to interfere with vocalization. In cases of septic crypts, the necessity for the removal of the entire tonsil cannot be questioned.

It is also important to consider the position of the tonsil and its relation to the surrounding parts when deciding upon the operation to be performed. In many cases hypertrophic tonsils are situated so superficially, although projecting into the throat, they do far less harm, except in a mechanical way, than the so-called submerged tonsils—those that are imbedded deeply between the pillars and so covered by them that free drainage from crypts is impossible. Whereas in the case of the superficially located tonsil drainage is free and unobstructed, consequently systemic infection does not readily take place, as it does in the case of the deep-seated tonsil with obstructed drainage; it is, therefore, the submerged tonsils that give rise to the most serious results. In many cases such tonsils remain entirely unsuspected, for the reason that the anterior pillar of the fauces so completely covers them that they cannot be seen upon ordinary inspection of the throat. It is only by drawing the pillars forward with a curved instrument so as to uncover these tonsils that their real condition can be discovered. In many cases this septic material remains pent up in the deeper crypts of the tonsils until it becomes a veritable abscess, and it is not an uncommon occurrence on removing such tonsils to find from half a drachm to a drachm of pus. This at once explains the relationship of rheumatism, arthritis, endocarditis, pericarditis, pneumonia and a host of other systemic infections from tonsillar infection to tonsillar disease, and to which they can be directly traced.

I quite agree with Dr. Casselberry that there are frequently other morbid conditions of the throat associated with diseased tonsils and at the same time requiring attention. The association of adenoids with diseased tonsils is generally understood, but there is often a chain of glands, extending from the base of the faucial tonsils to the lingual tonsil, that is very commonly overlooked, and unless these are removed at the same time that the faucial and lingual tonsils are removed much of the throat irritation, and particularly of the larynx, will continue, to the disappointment of the patient regarding the result of the operation.

Dr. George L. Richards, of Fall River: I performed a tonsillotomy on a young woman some ten or twelve years ago, and since that time she has been in perfectly good condition until recently, when she had a very marked peritonsillar abscess and nearly died. This was due to an infection through a small bit

of tonsil that had been left in the superior arch, and shows that after a tonsillotomy one is never safe from the danger of subsequent trouble having its source in the tonsil stump which remains.

A number of cases have been reported of infections which followed after tonsilleetomy, and, of eourse, this might be true during the time healing was taking place. I have had one case of purulent pleurisy coming on about five or six days after a tonsilleetomy. I operated on two sisters on the same day. One sister did very well, but the other, five or six days after, developed pleurisy, with effusion. I think in this case there was a direct infection or invasion through the open wound. I think nature is very kind in not permitting us to see such cases more often. Dr. Koplik has reported some time this last year cases of infection following operation, and these are conditions that really are often unavoidable because it is so difficult to keep the mouth wound aseptic.

Dr. Henry L. Wagner, of San Franciseo: In observing a large number of follicular tonsillitis cases the original seat of this infection is not to be found in the Waldeyer's Glandular Ring, but in the lower posterior portion of the ehoanal region; and in cases where tonsilleetomy has been performed usually a lateral or bilateral pharyngitis infectiosa has been observed. Systemic disturbances play a very important role in all these cases.

Dr. Albert C. Getchell, of Worcester: Until the bacteriology of the tonsils is definitely made out conelusions must be based on clinieal observations, and one case must be balanced against another. I will cite a case on the other side and speak of a possible danger coming from removal of the tonsils. I was called to see a patient supposed to have pleurisy, with effusion. The patient, a girl of nineteen, was seen in February. She had always been well up to the previous Summer, when she was operated upon for the removal of her tonsils, and had apparently made a good recovery from the operation. From that time on, however, her health had not been so good. Before I saw her she had been tapped, but no fluid was found. In my opinion she had a broncho-pneumonia. A day or two after I saw her, she had chills and a high temperature, and she went through a long period of siekness, expectorating foul, purulent matter apparently from an abseess of the lung. This was rather an extraordinary thing to happen in so young a person, and these sequelae, I think, might be dependent on a lowered resistance following and in a measure due to the tonsil operation.

Dr. Burt R. Shurly, of Detroit: The resistance of the patient and the bacterial findings are very uneertain quantities whieh

lead up to the difficulties of reaching any conclusions in any definite form. Throughout the various sections of the country it is interesting to note the wide difference of opinion on this very problem of operation. In our city of Detroit, for instance, we have a pediatrist, who absolutely demands that all tonsils be enucleated at the age of one year, irrespective of how much diseased. This man is simply an extremist. There are a large number of operators who enucleate the tonsils in every case that comes under observation, especially the young operators. It seems to me that the border line cases are the ones that give us so much trouble. What we decide to do with these cases is purely a matter of judgment.

It seems to me that this collective investigation that Dr. Clark has brought before us so ably shows a large number of cases of tuberculosis. It is claimed that a large number of infections occur through the tonsillar tract, and this is, in my opinion, one of the problems that should be added to this collective investigation.

This problem of tonsillectomy has been before this body and discussed for some years, and a great many have definite conclusions regarding it. It seems to me that so long as the physiology of the tonsils is so obscure we will naturally be at variance. It seems to me that the association should take up some research work on the problem of the physiology of the tonsils.

Dr. Lorenzo B. Lockard, of Denver: In some of these cases the electric cautery, which has just been severely condemned, is the method of choice. For a number of years I have employed it in certain cases, with satisfactory results. Many of my patients, in whom various systemic infections could be traced to the tonsils, have advanced tuberculosis, and are therefore not favorable cases for operative work. In these I have temporized by using the electric cautery, and when it has been thoroughly used the immediate effects, in many instances, are as favorable as after tonsillectomy. How long these effects will endure I am not in a position to say, but many have lasted for several years. One case in particular I may cite. This was the son of a prominent physician who had twice had his tonsils removed, but there were still some stubs containing a few crypts.

In the winter of 1911 he suffered an attack of acute articular rheumatism, with endocarditis. For three months he had fever and the swelling of the joints persisted.

At this time Dr. Lockard was asked to examine the tonsils, and cultures from the crypts showed streptococci. The crypts were cleansed and swabbed with iodine, and the temperature fell to normal, where it remained for several days. Each time

it recurred the same procedure was practised, with the same result. This was done a number of times, so that there could be no doubt as to the cause and effect.

The boy was flat on his back and his general condition precluded the possibility of radical operative work. He was, therefore, treated by electric cauterization of each crypt. So far there has been no recurrence of the rheumatism and the endocarditis has disappeared. In this case the procedure was certainly justified and successful.

I believe that while electric puncture cannot replace tonsillectomy as the method of choice, it has a distinct field of usefulness and should not be entirely abandoned.

Dr. William M. Ballenger, of Chicago: Speaking of focal infections, most physicians do not appreciate fully the significance of such infections. Three times I have been the subject of a focal infection, and know whereof I speak and what it means. Three times my life was actually in jeopardy from focal infection. Once from the intestines. Then I had a pyorrhea alveolaris of a marked degree. During the past winter I did not know what it was to be tired, whereas before I was tired all the time. As soon as I got rid of the focal infection of my gums this feeling went. Again last summer I was burned by one of our fellow members in the treatment of a corn, which was followed by a focal infection, with considerable danger to my life. These focal infections I have been able to overcome, and I now feel perfectly well and in better health than I have for years. So, therefore, we cannot afford to wait in these cases before undertaking the removal of the tonsils so soon as it is determined that they are the centres of infection. I have removed many tonsils in patients, but I cannot recall a case which made me realize that lost tonsils were essential to the patient as a functionating organ. It was thought that they produced some internal secretion. In many instances children from whom tonsils and adenoids have been removed become enormously fat and show changes that are not actually normal. In other cases perhaps there is left still a remnant or portion of the hypophyseal gland in the pharynx. Possibly I have never been convinced that the removal of the tonsils affects any internal secretion. To me it is merely a lymphatic gland and with no other function. If diseased I do not hesitate to remove it in its entirety. When the tonsils are to be removed because diseased I believe in their total, and not partial, removal.

I am much interested in what Dr. Makuen has stated regarding the conservation of the voice; at the same time I would rather have a live patient with a poor voice than a dead one with a good voice.

Dr. George A. Leland, of Boston: It seems strange that some surgeons report cases of acute middle ear disease after adenectomy, while others report cures of middle ear disease, both acute and chronic, by this operation.

In our volume of Transactions for 1909 are several papers bearing on this subject (notably Dr. Logan's), in which is strongly set forth the importance of clearing out the naso-pharynx, and especially the fossae of Rosenmuller, for the purpose of removal of infective foci and establishment of drainage and ventilation through the Eustachian tube.

It has been my custom, when possible, for twenty-five or more years to scrape out this fossa in inflammatory diseases of the middle ear, even in the acute cases in childhood, thereby obviating the necessity for paracentesis, almost (if not quite) invariably, even when this operation seems strongly indicated by red and bulging drumheads.

Dr. George E. Shambaugh, in closing: I will attempt to answer only those questions that have been raised in the discussion which relate to the subject of the tonsils as a focus for systemic infection. I am quite in accord with the remarks made by Dr. Ballenger. When one is dealing with a tonsil suspected of producing systemic infection one is not warranted in attempting any superficial temporizing measures. The one thing to aim at is to eradicate the focus of this infection and this is most readily accomplished by the complete enucleation of the tonsils.

Dr. Ingals: Are the objections raised against surface cauterization applied to the ignipuncture?

Dr. Shambaugh: My opinion is based upon experiences with cases that have been cauterized by other people, as I have not attempted cauterization for a number of years, due to several unsatisfactory results which I had a number of years ago. The patients have more tonsil trouble after the cauterization than before. I am inclined to believe that theoretically the cauterization of the crypts of the tonsils is open to the objection that it is impossible to cauterize to the bottom of these crypts, which run clear through the tonsil. As thorough a cauterization of these crypts as we are able to carry out may still leave infection in the base of the tonsil.

There are cases where with a high blood pressure and slow coagulation time enucleation of the tonsil is not to be undertaken. In these cases one must be content with other measures less radical, and here the cauterization of the tonsil crypts may be undertaken. I believe that as a rule the slitting of these pockets offers a better procedure than the cauterizing.

As regards the discussion bearing on the bacteriology of

the tonsils: there has been a great deal of superficial work attempted on this subject that has no value, as it has been undertaken by clinicians who are not trained bacteriologists. Dr. D. J. Davis and Dr. E. C. Rosenow, of Chicago, have in the last few years worked out pretty thoroughly the question of the bacteriology of the tonsils, and recently Dr. Rosenow has been able to isolate the particular type of streptococcus which is responsible for the joint trouble. This work has not yet been published.

As regards the question brought out in the discussion about the danger of producing a serious systemic infection when operating upon the tonsils: I believe that the tonsils that are acutely inflamed should never be operated. One should wait until the inflammation has completely subsided.

As regards the presence of a latent infection in a tonsil where the acute process has subsided, I have had the same experience which Dr. Coakley describes. Pus can often be expressed from these tonsils by exerting pressure over the anterior surface of the soft palate in such a way as to partially evaginate the tonsil.

I have had little experience in operating upon tonsils to improve the voice. I do not believe that the tonsil operation properly done is likely to have any bad effect upon the voice, but am very cautious about undertaking a tonsil enucleation for a patient with a trained musical voice. As regards the asymmetry of the soft palate after a tonsil operation, which Dr. Makuen laid so much stress upon, I do not see that this is a serious objection, since I have never seen a case where asymmetry of this sort produced any disturbance of the voice.

Dr. J. Payson Clark, of Boston: In closing the discussion in regard to what Dr. Makuen says about normal pillars, I do not see exactly what he means. I am sure that in the majority of cases operated on the pillars are perfectly normal so far as one can tell by inspection. They are clearly defined, perfectly symmetrical, and show no scar tissue, and appear to functionate as well as before the tonsils were removed.

With regard to what Dr. Casselberry has said about pharyngitis following tonsillectomy, my cases, so far as they go, show that such pharyngitis is not the result of tonsillectomy. I have always felt that an inflammation of the lateral pharyngeal folds is secondary to an infection from somewhere else.

With regard to what Dr. Coakley said about scarlet fever following tonsillectomy, my cases are too few in number to draw any conclusions. Dr. Edwin H. Place, physician-in-chief of the contagious department of the Boston City Hospital, is

taking records of the effect of tonsillectomy on susceptibility to scarlet fever, but these records are not yet available.

Dr. Logan has misunderstood me; in my paper I stated that only fifteen (15) patients had the tonsils removed without having any reason for their removal definitely stated.

I believe that the middle ear cases, five (5) in number, might have been due to incomplete adenoid operations.

In regard to the possible hypertrophy of other lymphatic tissues in the throat following tonsillectomy, I have observed none, although I did not make any definite search for it.

THE SIGNIFICANCE OF PLASMA CELLS IN THE TONSIL.

A Preliminary Report.

BY J. GORDON WILSON, M.A., M.B.

In an article published last year (The Tonsils in Childhood, American Journal of Diseases of Children, May, 1912, Vol. 3, pp. 277-286), I emphasized the significance of what I called the hyperfunctionating tonsil in childhood. I stated that "Mere fluctuation in size of a tonsil in a child ought not to be regarded as a sign of local disease, and an indication for enucleation. Many children are constitutionally subject to such fluctuations and the treatment is general more than local." "It is common knowledge that in systemic diseases we have such enlargements and few of us are disposed to argue that these are a sign of focal disease. I would rather regard them as indications of functional activity." Speaking with reference to the region where the respiratory and alimentary tracts meet I said "As knowledge advances there is constantly impressed on us the important and unexpected relation of this region to disease. We err if we forget that here as elsewhere the essential resisting power to infection lies in the tissue cells. Further, we shall add to this error if we concentrate our attention too much on leucocyte invasion or exudation to explain initial protection. Whenever from unhygienic surroundings or mode of life (it may be also from constitutional weakness) these tracts are exposed to influences which lower their vitality, the tonsils suffer for them and with them. When the mucous surfaces, particularly absorbent in the child, are exposed to irritation, the tonsil again suffers for them and with them. It seems to me the tonsils in a child protect these tracts, especially where they come together in the nasopharynx and are associated directly with

their well-being. If this be so, I am called on to do everything to protect these bodies during the period of functional activity."

An article which has had considerable influence on the question of the function of tonsils and on the importance of tonsillar disease in systemic affections appeared recently in the Journal of Infectious Diseases, by Dr. D. J. Davis (Davis, D. J.: Plasma Cells in the Tonsils, Journal of Infectious Diseases, March, 1912, pp. 146 and 158). In order to make Dr. Davis's opinion clear I should like to quote at some length from his article. He says (loc. cit., p. 158): "In all tonsils from individuals who are more than a few weeks old plasma cells are found in abundance. They occur under the epithelium, especially that lining the crypts, along the connective tissue bundles and about the blood vessels. In the hypertrophied tonsils they are much more numerous. Their significance is not entirely understood, but inasmuch as they are more or less characteristic of chronic inflammatory processes they probably indicate a chronic infection of the tonsillar crypts or the absorption of toxic substances *therefrom.*" In another part of the same article (loc. cit., p. 146), he says: "In view of the fact that plasma cells occur in such large numbers in tonsils, appendix, and some other tissues after birth at a time when the crypts and intestinal canal are infected, and associating this with the fact that in chronic infections generally the plasma cell is so constantly found one is led to suggest that their presence in the tonsil and appendix, and also in the gastro-intestinal tract, lymph glands and spleen is due to a chronic infectious or absorptive process." The suggestion of Dantchakow and Pirone, that the absorption of ferments may cause sufficient irritation to promote the accumulation of plasma cells, is passed over by Dr. Davis with the remark that "In the case of the tonsil, the view is more rational that this organ is in all cases, whether hypertrophied or not, a chronically infected focus shortly after birth, a view apparently supported by bacteriological studies. It therefore seems reasonable that cells more or less characteristic of chronic inflammation should accumulate here. Not only the nature of

these cells, but their accumulation in such large numbers and in localities immediately underneath the epithelium, especially about the infected crypts, also favors such a view."

The views here expressed by so competent an observer as Dr. Davis appeared to me to be somewhat at variance with what I had written. I therefore determined to inquire into this aspect of the subject. I was further impelled to do so since, if his conclusions are correct and the opinion here expressed of the significance of plasma cells in the tonsil be true, we seem driven to the conclusion that the supporters of complete enucleation of the tonsil under all conditions have proved their contention that the tonsil is from the earliest stages of life a diseased organ which it is our duty to remove. The conclusions that may be drawn are perhaps wider than Dr. Davis himself will admit. For it appears to me we must further admit that the tonsils and the appendix are vestigial structures. So far as the appendix is concerned we need not here concern ourselves except to note that competent anatomists and physiologists and some very excellent surgeons are not so sure as they were a few years ago that it is a vestigial organ. We are, however, concerned with the tonsil, and again I reiterate what I have already frequently declared, that neither embryology nor comparative anatomy nor physiology gives any support to such an hypothesis. The opinion that they are vestigial in man and even worse than useless has done considerable damage and created prejudice against much endeavor to discover their nature and function.

Every one is becoming more and more convinced of the great importance of the lymphatics and of the adenoid tissue of the pharynx. But the opinions held of the role they play in regard to absorption of micro-organisms and toxins are very divergent, though the current view would appear to condemn the large masses of adenoid tissue root and branch. Though much work is being done, much more requires to be done before we can have any but the haziest ideas about their significance. However, if we start any scientific investigation bearing on this question with a false assumption in regard to

tonsils and adenoid tissue generally, our results must be hopelessly astray. It is well therefore that we at times pause to examine the basis for our theories.

Plasma cells occur mostly after birth in blood forming organs (Maximoff, Fol. Haem., 1907, Bd. IV., p. 625). They arise under certain conditions from two allied groups of cells—lymphoblasts and lymphocytes—and are best designated as lymphoblastic or lymphocytic plasma cells. It is of the utmost importance to remember the relation of the lymphoblasts to the lymphocytes. These cells differ morphologically only in size. (Aschoff: Pathologische Anatomie, Bd. 2, p. 102.) Lymphoblasts are cells of the size of myelocytes which possess a weak basophile protoplasm. The fairly large, somewhat irregular nucleus shows a relatively thick cell membrane and strongly colored nuclear granules. Close to the nucleus, usually to one side, there is in the protoplasm a small clear halo which represents the position of the red granules of the Altmann-Schridde stain. The lymphoblasts, according to Aschoff, are a division stage of the lymphocyte and may show mitosis. From them there again arise lymphocytes, small cells with a round chromatin rich nucleus, which usually lodge nuclear bodies which stain strongly. Also around these there is the clear halo comparable to that of the lymphoblasts.

The lymphoblastic plasma cells are less frequently observed than the lymphocytic plasma cells. The former possess the typical lymphoblast nucleus, but the protoplasm is more strongly basophile and the halo is larger. The lymphocyte plasma cell has a pronounced nucleus which lies at one end of an almost oval cell. The halo and Altmann-Schridde granules are found in both.

The Unna-Pappenheim *Pyronin-Methylgreen* stain which I have chiefly used stains the cytoplasm red and the nucleus blue green; the characteristic features of the plasma cells are the eccentrically placed "wheel" nucleus, with the small clear halo around it. It is called a wheel nucleus because the chromatin usually radiates centripetally. Plasma cells may show mitosis as well as two or three nuclei (Pappenheim Fol. Haem., 1907, Bd. IV., p. 206).

Plasma cells are acknowledged to be derived from lymphocytes by irritative processes. In artificially produced inflammation due to toxins, plasma cells appear in great abundance, and certain toxins have a special tendency to produce them. They are found in the vicinity of sterile foreign bodies; e. g., sterile sponges. Their abundance in chronic infectious processes has made such processes a favorite *locus* for the study of plasma cells. From this has developed the predominating idea that these cells must be necessarily connected with inflammatory processes.

One great objection that can be raised in regard to many of the investigations into their function is that they have in the main been studied in pathological tissues, and so have come to be regarded more from their pathological than their physiological side. Hence has come the undue emphasis which has been placed on their degenerative characteristics. Unna and many pathologists regard them entirely as degenerated cells. It would appear, however, from further investigations that this is too narrow a view, and a view which in the tonsil will be fraught with unfortunate consequences.

It is well recognized that plasma cells occur in all subacute and chronic infectious diseases; but it is not so often remembered that they are also found in organs which we are justified in believing are normal, of course never in such abundance. Thus plasma cells are found in man and other mammals in the hemopoietic and lymphatic systems and in the digestive tract with its associated glands. Professor Schaffer (Schaffer: Centralblatt f. Allgem. Path., 1909, Bd. XX, p. 1910) gives a list of various organs where plasma cells are normally found. He sums up as follows: "In short, wherever lymphocytes are, plasma cells can be met with. It is especially to be noted that in many of these places, in particular physiological conditions (digestion, pregnancy) the number of plasma cells can be increased enormously."

There are many facts which compel us to recognize that they are engaged in many normal activities. Thus they appear to be necessary to the discharge of such physiological

activities as digestion and of repair. Pirone (Pirone: Folia Haemat. 1909, Bd. VII, p. 342) demonstrated during digestion a marked increase of plasma cells in the intestine and spleen. This was confirmed by the observation of Joannovics (Joannovics: Centralblatt f. Allgemeine Pathol. und Pathol. Anatom., Bd. XX., p. 1222) that dogs seven hours after a test meal showed in the intestinal mucosa and submucosa enormous numbers of plasma cells, which lie partly scattered throughout the tissues and partly gathered into clumps. Here they may often be seen with many nuclei quite similar to giant cells. The increase of plasma cells in the spleen during digestion is mostly in the pulp, and here they are plentifully found. Very interesting is the interpretation of Dantchakow (Joannovics, loc. cit., p. 1021), who concluded from the results of her experiments that there is a connection between glandular activity and plasma cell formation. She attributes to them the function of taking up and carrying away materials elaborated by secretion.

When we compare the results of these experiments with the examination of plasma cells both in normal and pathological conditions, and when we find that they are found physiologically in the lymphatic tissues and also in the digestive tract with its related glands, the suspicion is immediately awakened that probably the cause of plasma cell production may be referred to some irritant resulting from glandular activity or from the action of ferment as well as from cell degeneration and increased protoplasm destruction. In agreement with this view we have the experiments of Foa, who produced abundant plasma cell formation by injecting organ macerations and by inserting foreign muscle tissue. The irritative action of such ferment-like products will explain not only the appearance of plasma cells normally during digestion and in atrophic processes, but also the physiological appearance during involution of the thymus and as a sign of senescence of bone marrow.

What appears to me to be a clear conception of their function was enunciated by Loewitz (Loewitz: Centralblatt f. Allgemeine Pathologie und Pathol. Anatomie, Bd. XX, No. 22, p. 1030), who declares that he could not regard the plasma cells as

degenerated elements, for when they finally degenerated this followed after the exercise of a certain function. He believed that not only could their formation be caused by ferments, but that they are themselves necessary to produce ferments and so are actively engaged in the digestion of albuminous bodies. This is well in keeping with the view of Schaffer (loc. cit., p. 1010): "Die Funktion der Plasmazellen besteht in der Fortschaffung and Nutzbarmachung zerfallenden Zellmaterials." Joannovics (Joannovics: Centralblatt f. Allgemeine Pathol., 1909, Bd. XX, p. 1025), in concluding an exhaustive article on this subject, declares that in all likelihood they partly represent specific transformed elements with less functionating power. Sooner or later they perish by vacuolization, liquefaction or hyaline degeneration with pyknosis of the nuclei. In regard to their origin, the active cause seems to be the irritative action of a hypothetical ferment-like substance produced by secretory glandular activity, by atrophic conditions and by the disintegration of cellular elements.

We are here chiefly concerned with the significance of plasma cells in adenoid tissue. This tissue has, of course, been chiefly studied in the follicles of lymph nodes, where, as has been said, plasma cells are normally present in considerable numbers. As is well known, adenoid tissue is the seat of the exchange of materials between the blood and the lymph. It has been shown that the lymph contains specific substances that do not exist in arterial and venous blood. Many of the katabolic products poured out of the tissues into the lymph stream if directly reabsorbed into the blood would exercise a toxic action. "These products are rendered innocuous and even beneficial to the organism by the specific activity of the lymph nodes through which they pass before re-entering the blood" (Luciani's Physiology, Vol. 1, p. 534, translated by Welby, London, 1911). They are destroyed and modified in the adenoid tissue. It would appear, therefore, that adenoid tissue has a more important function than that of filters to arrest or retard the entrance into the blood of mechanical impurities, be they particles of dust or pathogenic organisms.

One of their physiological functions is to destroy the toxins of tissue metabolism.

What part the lymphocyte and its derivative the plasma cells take in this function we do not know. We do know that as the toxins increase so also do these cells. Luciani believes that their destruction is a direct consequence of the metabolism of the lymphocytes accumulated there in large quantities. The results of various investigators support the view that the lymphocytes are actively engaged and that in the battle plasma cell formation occurs and as a sequence many plasma cells degenerate. In the tonsil plasma cells are normally present, but their presence is no more an indication of chronic local disease than their presence in lymph nodes. On the other hand, it would be a sign of disease or, at any rate, of a non-functionating organ, were they absent.

The tonsils I am here concerned with have been hypertrophic and have been obtained from children and young adults. In all the adenoid tissue was abundant and both lymphocytes and plasma cells were in excess. In many cases increase of the plasma cells was out of all proportion to the increase of lymphocytes. This increase indicated that the lymphocytes are here more actively engaged, and that some injurious body is present in excess. Does this local increase in number of plasma cells give any indication as to the source of the irritant? We may presume that the irritant may come from the crypts or through the lymph channels; may come from focal disease or from some distant katabolic process.

In most of the specimens of hypertrophied tonsils plasma cells are more abundant near the crypts. But they are not confined to the subepithelial region bounding the crypts. They can be found in the follicles in the connective tissue between the follicles and in the deeper parts of the tonsil. Davis says that he has been unable to find plasma cells in the follicles. I will show you some specimens of hypertrophied tonsils in which they are present. Many of these in the centre are large and belong to what Schridde has called lymphoblastic plasma cells, but both in the centre and towards the periphery of the follicle you will note many typical plasma cells. In some of

the slides you will note the plasma cells not only at the part of the follicle towards the crypt, but also in the outer zone remote from the crypt. In what appeared macroscopically to be a perfectly normal tonsil of a monkey, the most abundant plasma cells are found in the deeper part of the tonsil; and in the connective tissue were more abundant than near the crypts. In some of these slides it would appear as if plasma cells near the crypts are more abundant at the point of exudation of the leucocytes where Stohr phenomenon is taking place.

From the sections I have examined there is evidence that certainly not all of the irritants that stimulated the plasma cell formation have entered from the crypts. There is evidence that irritants may come through the lymph stream from extra tonsillar areas. But it matters not how or from where the irritant comes the plasma cells are there to meet it. In the tonsil the plasma cells indicate not so much a diseased organ as an organ that is reacting to disease. Their localization indicates not only that there is absorption of toxins from the crypts, but that there are toxins coming from other sources. Their early occurrence is no argument for early removal, but rather for the putting right of any faulty metabolism which has caused their increase. It calls for less concentration of thought on a narrow field and a wider range of view. By doing so we will have advanced preventive medicine and added to the well-being of our patients.

The presence of plasma cells instead of showing that the tonsil ought thereby to be regarded as a source of danger would rather indicate an organ which is striving to preserve the body from disease. Their presence in excess certainly indicates that something is wrong, but not necessarily in the organ itself. The tonsil is diseased not because plasma cells are present, but because they are no longer able to cope with the toxins pouring in.

The important function of the tonsil is to protect the body from poisons absorbed from the pharynx, probably also as Metchnikoff believes (Metchnikoff, Immunity in Infective Diseases, p. 429) to pour into the pharynx large numbers of lymphocytes which act as a defence against organisms in

the pharynx. If they are diseased and non-functionating they are worse than useless because they allow the passage of these poisons. I can well imagine that their removal may cause the lymph stream to be diverted to more active and better functionating tissue.

Of course we have diseased tonsils just as we have diseased lymph nodes. Of course we have to enucleate tonsils just as we have to enucleate lymph nodes. I am not advocating the preservation of a diseased organ. I am pleading for a scientific basis for our knowledge when the tonsil is diseased. I am pleading for a recognition of its function and a more just conception of how a faulty metabolism here—from its size, its position and its anatomical structure—must have a far reaching effect. The duty of the laryngologist is not that of a mechanic to remove the diseased organ. He should strive to prevent the organ from becoming diseased; he should be prepared to correct the faulty metabolism which may have led to the disease. The objection to specialism that its view of disease is too circumscribed, can well be urged in regard to our attitude in tonsillar treatment.

In conclusion I would sum up my position thus: Plasma cells are derived from lymphocytes and are engaged in removing and utilizing cell material which has broken down. If it be pathological to destroy albuminous bodies and toxins arising from katobolic processes—and possibly also of bacterial origin—then plasma cells are pathological. They are not degenerated cells, but cells actively engaged in combating the toxins which pass through adenoid tissue. Their presence, instead of showing that the tonsil is diseased, indicates that it is functionally active. Their presence in excess shows that we have some focus of disease, but not necessarily a local one.

Discussion.

Dr. Algernon Coolidge, of Boston: I have recently studied the question of the normal size of the tonsil in childhood. While in adults tonsils vary much in size in different persons, in children they vary surprisingly little. In a series of 200 tonsillectomies in children, those tonsils which have previously been

diagnosed as large or hypertrophied by different physicians weigh on an average less than those which have been called small, but are removed for constitutional reasons. The apparent difference in size is often entirely due to the varying relation of the tonsil to the soft palate. In a child of seven it is very unusual to find a tonsil of less than thirty or more than fifty grains.

Dr. Henry L. Swain, of New Haven: The association is to be congratulated upon having had presented to it such an excellent paper. I think that work of this kind will stimulate us to further inquire into the physiology of the tonsil and estimate what its actual function is. I am not clear in my mind as to the nature and function of these plasma cells and welcome any further elucidation.

Dr. Henry L. Wagner, of San Francisco: I have been interested in this paper; that these cells in their activity might be compared with the ciliated epithelial cells in various parts of the respiratory tract; that in normal condition of our body these cells take up a natural defense against invading microorganisms and if impaired infection then may take place.

Dr. George B. Wood, of Philadelphia: I am very much interested in the subject under discussion and have noted an almost constant occurrence of plasma cells in the tonsils. It is generally accepted to-day that the presence of plasma cells indicates a condition of sub-acute inflammation. I have noticed, especially from my studies in connection with tuberculosis, that though the tonsil is readily invaded by bacteria, there is a distinct resistance of the tonsil parenchyma to the action of bacterial toxins; for instance, an infection of the tonsillar tissues with tubercle bacilli rarely produces progressive lesions, more frequently it is found as a latent type with a tendency toward fibrosis of the tubercles. Should, however, these bacilli infecting the tonsil gain access to the efferent lymphatics and invade the regionary lymph nodes, there follows a rapid destruction of these lymph nodes out of proportion to the progress of the disease in the tonsil. This has been demonstrated experimentally and confirmed by clinical observations. Now presence of plasma cells indicating a mild degree of inflammatory reaction are evidence that the tonsils are being subjected to frequent insults from bacterial invasion, and that they have reacted with the production of a localized immunity.

TONSILLAR INFECTION.

*A Preliminary Report Concerning the Passage of Anthrax Bacilli Through the Tonsillar Tissues as Determined by Experimental Research.**

BY GEORGE B. WOOD, M.D.

The determination of the importance of the tonsils as a gateway for cryptogenic infections is one of the most difficult problems in the domain of the laryngologist. Notwithstanding certain sporadic outbursts of conservatism, both from specialists of repute and from those who by striking fire desire to gain fame, the progressive clinicians are becoming more and more convinced that the tonsillar masses of the throat are responsible for a large number of general systemic infections. It takes a long time, careful observation and many reconsiderations to arrive at the truth of a difficult subject through clinical study alone, hence the necessity of resource to animal experimentation. The disadvantages, however, of this method are manifold, especially so when we come to study tonsillar tissues. Practically the only available animal which has a tonsillar structure sufficiently large and easy of approach for study is the hog, but unfortunately the hog is one of the most resistant of animals to the invasion of micro-organisms. There are, however, certain diseases to which this animal is subject, and it was thought that by using the specific organism of one of these diseases the importance of the tonsillar tissues as a gateway of infection might be studied experimentally.

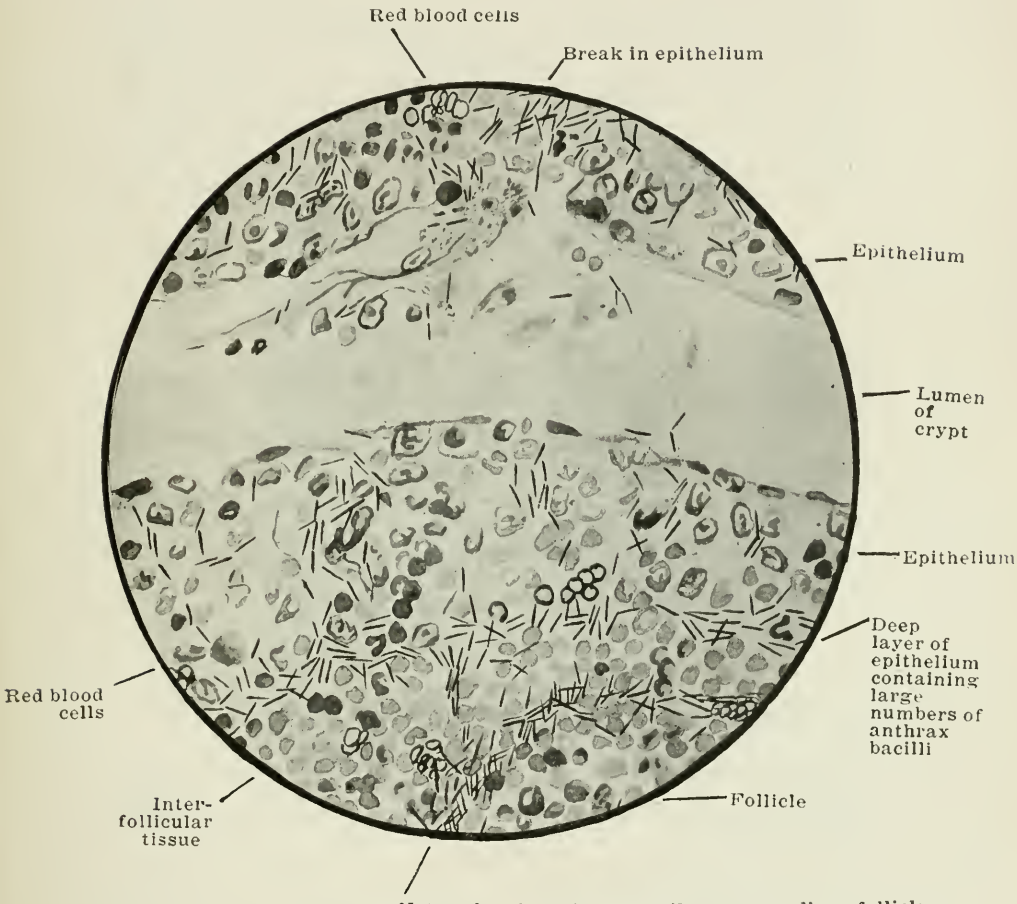
In 1904 I published the results of a series of experiments on tuberculosis. In those experiments I confirmed certain results,

*I herewith beg to express my deep appreciation of the courtesy extended by the members of the John Herr Musser Department of Research Medicine of the University of Pennsylvania, and of the Laboratory of the Pennsylvania Livestock Sanitary Board, whose kind assistance and co-operation were such as to enable me to get distinct results.

previously obtained by Ravenelle, showing that the tonsils of the hog are readily inoculated without trauma by simply bringing the tubercle bacilli in contact with the mouths of the crypts. Ravenelle found that the tonsils were always infected where tubercle bacilli were present in the food. In my own work I rubbed a swab saturated with an emulsion of tubercle bacilli over the tonsils and always obtained a successful inoculation. I might state that in no instance was any other portion of the pharyngeal mucosa involved. As far as tuberculosis is concerned these results have been verified by the clinical and postmortem examinations of human tuberculous subjects. It is safe to say that at least 90 per cent of all people dying from pulmonary tuberculosis show secondary involvement of the tonsils, and that, as shown by a collective study, the average proportion of primary tuberculosis of the tonsils in children is from four to five per cent of all tonsils removed by operation.

These results, however, do not warrant the assumption that the tonsils are open to a similar invasion from other organisms and especially from those causing acute infections. In our study of tonsillar invasion we must remember that only pathogenic bacteria can be considered, and in experimentation it is frequently found that bacteria pathogenic to man are not so to animals. For instance, a strain of streptococci obtained from a case of acute rheumatic arthritis was injected directly into the blood of the hog. This was done to find out whether lesions similar to those in man could be reproduced in a hog, and if such lesions developed an effort was to be made to determine the possibility of the tonsils acting as a portal of entry. Unfortunately for the experiments, the inoculated hog flourished and grew fat under doses that would have proved fatal to almost any other animal. It was, therefore, necessary to choose an organism of known pathogenicity to the hog. The two which seemed most likely to afford results were the anthrax bacillus and the bacillus of hog erysipelas. The former was easily obtained and possessed certain advantages, namely, its ready staining in tissues and its easy culture on ordinary media, and was therefore selected for the work outlined in this paper.

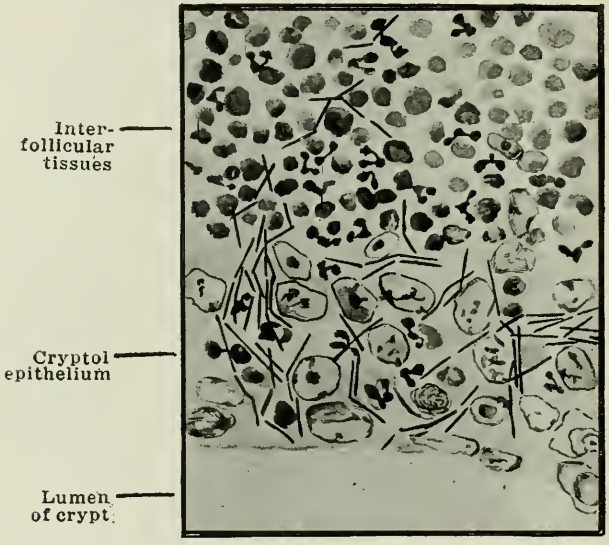
Anthrax is a contagious disease commonly found amongst the domestic animals, frequently appearing in epidemics. It



Section from the tonsil of Hog No. 5, showing penetration of the anthrax bacilli through living epithelium; a tendency to multiply in the deeper layers of the epithelium and a congregating of the bacilli in a dense network around the periphery of a follicle; also a gap in the cryptol epithelium, probably due to the toxic action of the bacilli Free red blood cells can also be seen in the tonsil parenchyma. Some of the bacilli free in the crypt lumen show a capsule.

Section stained with Borax Carmine and Gramm's Method.

occurs sporadically amongst hogs, but never in the epidemic form, and it has been shown that certain breeds of hogs are more resistant to the invasion than are other species; also the adult animal is more resistant than the young, and it is probable that individuals show varying degrees of susceptibility. Among hogs pharyngeal anthrax is by far the most frequent variety. Occasionally the source of infection is through the intestine and then the symptoms correspond to inflammatory conditions of these organs. The course of the disease is very



Section through edge of a crypt from the tonsil of Hog No. 2, showing anthrax bacilli passing between the epithelial cells. Cryptol epithelium unaltered except for the presence of polymorpho nuclear leucocytes.

rapid, and the animals generally die in from one to two days, though sometimes they will live several days or a week. In pharyngeal anthrax the portal of entrance is always the tonsils. It has been reported that the base of the tongue and the pharynx are occasionally the atrium of infection, but there is no proof that such is ever the case. The diseased area first shows swelling and hyperemia and later necrosis with hemorrhagic infiltration. The necrotic material is either brownish or

yellowish gray in color and the cut surface cloudy and relatively dry. It is firmly attached to the underlying tissues. The neighboring mucous membrane of the palate and the tongue, the pharynx and the larynx, is generally swollen and reddened, and may show small petechial spots.

Occasionally small carbuncles form on the mucosa, which may later break and form ulcers. The disease spreads rapidly into the neighboring connective tissue, not only that underneath the mucous membrane, but also that between the muscles and beneath the skin, and gives rise to extensive edematous infiltration.

The regionary lymph glands show peculiar changes; they become enlarged and are dark brown red in color, sometimes uniformly so, and sometimes blotched. On section through the brown red areas the cut surface is not smooth but finely granulated and cloudy. The nodes generally infected are the submaxillary, the sub-parotid, and the upper cervical.

Pathologic Histology.—Wherever the anthrax bacillus accumulates hyperemia is rapidly produced, followed by exudation of blood serum and leucocytes. In the sub-acute cases, there may also be proliferation of the existing lymphocytes. This condition is soon followed by an extensive extravasation of the red blood cells and tissue necrosis. It is characteristic of anthrax that the nuclei of the cells do not, as is usually the case in other forms of necrosis, at first show an increased staining reaction, but that from the beginning the nuclei become more and more shadowy, and finally, after they are broken up into small indistinguishable particles, entirely disappear. The cell body generally becomes invisible before the nuclei, and the erythrocytes retain their staining qualities the longest. The bacilli enter the lymph spaces and pass through the lymph vessels to the regionary lymph nodes. In the lymph glands they accumulate in the perifollicular sinuses and are found only in small numbers in the lymph follicles themselves.

Experiments.—The first two hogs used in these experiments had previously been inoculated into an ear vein with a strain of streptococci, taken from a case of acute articular

rheumatism. This was done to determine the susceptibility of the hog to rheumatic infection. There was, however, absolutely no reaction either locally or general.

Hog No. 1.

Animal weighed about 50 pounds, and was in good condition.

March 14.—The surface of the tonsils, especially the right, was rubbed over with a 24 hr. culture of virulent anthrax bacilli. (The culture had not been previously emulsified.)

March 15.—Animal killed with chloroform. Cultures were made from the pre-scapular, sub-maxillary, sub-parotid, and retro-pharyngeal lymph nodes; also from cut surfaces of each tonsil. The tonsils were removed and kept for histologic study. Except for a slight congestion of a small part of the right tonsil, there were no gross lesions.

All the cultures from the lymph nodes, after having been incubated for two days, remained sterile. Cultures from the tonsils showed staphylococci and other organisms, but no anthrax.

Histologic examination of the tonsils showed normal tonsil structure, except that a few of the crypts possessed an excess of polymorphonuclear leucocytes. In a very few crypts staphylococci were found, but no anthrax bacilli could be seen anywhere.

Comment.—This was a negative experiment and a possible explanation of the failure of inoculation was that the bacilli were not first sufficiently separated before they were applied to the surface of the tonsil.

Hog No. 2.

Animal weighed about fifty pounds, and in good condition.

March 18.—Tonsils were swabbed with an emulsion of anthrax bacilli, most of it being rubbed into the right tonsil.

March 20.—Animal killed with chloroform.

Cultures were made on agar from both tonsils, the left sub-parotid lymph node, the left retro-pharyngeal lymph node and the left pre-scapular lymph node, the right retro-pharyngeal

lymph node, the bronchial lymph nodes, the lung, the spleen, liver and kidney. Bouillon cultures were made from the blood, stomach, large intestine and small intestine.

The tonsils were flecked with fibro-purulent exudate, apparently coming from the mouths of the crypts. On the left tonsil there was a coalesced area involving about one-quarter of the tonsillar surface. The tonsils were slightly swollen and protruding on the surface. There was no distinct congestion to be seen from surface examination, but on section there were definite inflammatory striae running through the parenchyma from the surface toward the base. A few small abscesses were found in the tonsillar substance.

Cervical lymph nodes were normal.

The lung showed scattered throughout its substance a number of circumscribed, congested and slightly edematous atelectatic areas. These were more numerous in the right than in the left.

Bronchial lymph nodes were normal.

Liver was normal.

Spleen was normal.

Kidneys were normal.

The cultures from the right tonsil showed staphylococci and anthrax; from the left tonsil, anthrax. There was no growth on cultures from any of the other organs, nor from the blood and gastro-intestinal tract.

Histologic Examination.

Both tonsils showed somewhat similar lesions though the infection was very irregularly distributed. While the greater part of the tonsillar structure was normal, almost all of the crypts contained an unusual amount of cellular debris, polymorphonuclear cells being predominate. There were, however, some lymphoid cells and quite a large number of epithelial cells. In a few crypts staphylococci were present in large numbers and a few anthrax bacilli could be seen. The large majority, however, of the crypts contained no visible organism, even where the surrounding walls and tonsil paren-

chyma were extensively invaded. On the surface of the tonsil the predominate organisms were cocci, generally, staphylococci, a few small bacilli and an occasional anthrax bacillus. The surface epithelium was generally intact, though in places it was covered by a layer of necrotic tissue containing large numbers of polymorphonuclear cells and necrotic superficial epithelial cells, being more numerous in the deeper layers. This change in the surface epithelium was most marked near the crypt openings.

Around the infected crypts, the greater portion of the epithelium was either entirely destroyed, so that only a few scattered necrotic cells were visible, or were so infiltrated with polymorphonuclear cells and lymphocytes as to make its outline scarcely distinguishable. There was, however, normal epithelium around a part of the crypt. In some places large numbers of anthrax bacilli could be seen between unaltered epithelial cells, being more numerous in the deeper layers. Near these places, that is, where there was anthrax bacilli in healthy epithelium, the neighboring interfollicular tissue showed only a few of the anthrax bacilli. The bacilli seemed to be passing in the direction of the lymph current, and in places the lymphatic spaces and some of the smaller efferent lymph vessels were choked with a dense network.

Another important finding was that anthrax bacilli tended to accumulate around the periphery of the lymph follicles, forming in places a dense ring around the edges of the follicle with only a few scattered bacilli in its centre or in the surrounding interfollicular tissue. In the neighborhood of those crypts of which the epithelium had been destroyed, the interfollicular tissue and also the follicles, showed the presence of numerous polymorphonuclear cells, congested capillaries, fragmentation of both the lymphocytes and other cells, and quite a large number of free erythrocytes. Where the necrotic action was more marked all cellular contour had disappeared and the space filled with a striated non-staining debris. Anthrax bacilli were present in this necrotic tissue, seldom, however, accumulating in any great numbers. In a few places associated with the anthrax large numbers of staphylococci were

found. Some of the anthrax bacilli situated in the unstaining necrotic tissue showed plasmolysis, otherwise these bacilli stained regularly and evenly.

The histological examination of the lungs showed that in the atelectatic area the alveoli contained a considerable amount of free red blood cells.

Otherwise the lung tissue was normal.

Hog No. 2.

Comment.—The most interesting finding in this experiment was that the anthrax bacilli passed through normal epithelium, showing a tendency to multiply in its deeper layers. In the neighborhood of this invasion there were comparatively few bacilli in the interfollicular tissue, while around the neighboring lymph-follicles and in some of the mouths of the lympathics large dense masses of anthrax bacilli were seen. Also, the disease was entirely localized in the tonsils, there being no lesion in any other organ, nor could cultures reveal the presence of anthrax bacilli. Their absence from the gastro-intestinal tract must mean that in pharyngeal inoculation if the anthrax bacilli do not soon find a point of invasion they are swept away and destroyed by some agency in the gastro-intestinal tract.

Hog No. 3.

March 26.—Animal about fifty pounds in weight was infected with sporothrix and was also suffering from a traumatic myelitis.

A 24-hour culture of virulent anthrax rubbed over tonsils.

March 29.—Animal killed with chloroform. Post mortem examination showed no signs of anthrax either in the tonsils or in other portions of the body.

Comment.—This hog was an adult animal, and it may be that it had developed immunity to anthrax infection some time during its life.

Hog No. 4.

April 16.—A 24-hour culture of anthrax bacilli rubbed into the left tonsil.

April 19.—Animal killed with chloroform.

Post Mortem.

Tonsils.—On the left tonsil there were three or four elevated areas with a central necrotic core surrounding which was a zone of hyperemia; right tonsil, only one small area as described above. The mouth, pharynx, larynx, trachea and oesophagus showed perfectly normal mucosa. There was no enlargement of the lymph nodes of the neck, and the lungs, liver and spleen were normal. A bouillon culture was made from the heart blood and agar cultures from the left submaxillary, left sub parotid and the right sub parotid lymph nodes, also from the lungs, spleen and liver.

April 21.—All the cultures remained sterile.

Histologic Examination of the Tonsils.

There was apparently no involvement of the tonsillar tissue except the small carbuncle. At this point the disease penetrated deeply into the parenchyma and consisted of a necrotic core surrounded by a thick zone of inflammatory reaction. The area of disease corresponded in size and extent to a large crypt, of course, involving the neighboring parenchyma. Portions of the crypt wall were still found intact with normal epithelium, especially at its deep extremity. The surface epithelium covering the diseased area was necrotic, and the superficial layers were interspersed with numerous staphylococci. The anthrax bacilli were distributed throughout the diseased area, but were more numerous in the living tissue outside of the necrotic core. They were not found in the normal tonsil structure. There was a secondary invasion with staphylococci, and in one place there was a beginning abscess near the capsule of the tonsil due to this staphylococci invasion. The staphylococci could also be found penetrating into the lymph radicals and accumulating on the periphery of one or two lymph-follicles. The staphylococci had gained entrance to the parenchyma of the tonsil through an epithelial gap in the walls of the crypts. Just how these gaps in the epithelium had been formed could not be determined, but a few staphylococci were found free in these gaps, and quite a

number in polymorphonuclear phagocytes in the parenchyma of the tonsil near this opening. The most resistant tissue to the action of the anthrax toxines were the germinating follicles, and these structures could be seen almost normal where the surrounding parenchyma had been entirely destroyed. The blood vessels also showed great resistance, and were more numerous and larger than in the normal tonsil structure. The anthrax bacilli accumulated in large numbers around some of them and occasionally were found actually penetrating their walls, entering the blood current.

Comment.—It is probable that the original invasion in this case was due to anthrax, and that there was also a secondary invasion of staphylococci. The staphylococci were enabled to gain access to the parenchyma of the tonsil due to the action of the anthrax toxine. The disease had advanced beyond a stage in which the anthrax bacilli could be seen in the living cryptal epithelium, consequently the mode of invasion could not be determined by this experiment alone.

Hog No. 5.

April 24, 1913.—Adult animal weighing about 70 lbs. Rubbed an attenuated (at 42.50C for 12 days) 24-hour culture of anthrax (Vaccine strain B) over left tonsil.

April 28, 1913.—Animal killed with chloroform. Heart puncture made and blood taken into a bouillon tube for culture. On the right one there was a small carbuncle and a small congested area, while the left tonsil was apparently normal. There was no lesion in any other part of the pharyngeal mucosa or in the oesophagus, larynx or trachea. Cultures were made from the right sub-maxillary and retro-pharyngeal lymph nodes.

April 30, 1913.—All cultures were sterile.

Histologic Examination of the Tonsils.

The tonsil in the region of the carbuncle showed very interesting changes. The carbuncle consisted of a central necrotic core surrounded by a distinct inflammatory zone. In

the necrotic tissue large numbers of staphylococci were found and in some places they were seen penetrating into the living tissue of the inflammatory zone. Only a very few anthrax bacilli could be found in the central part of the necrotic area, though they were fairly numerous around its edges. On the other hand in the zone of inflammation, large numbers of anthrax bacilli were found. At places they accumulated, forming dense net works, and also showed a tendency to gather around the periphery of the germinating follicles.

The diseased area was more or less associated in its topography with the position of a crypt, although it had, of course, advanced beyond the cryptal lumen and involved the surrounding tonsillar parenchyma. In some places the relation of the secondary infection of the staphylococci to the invading anthrax bacilli could be traced. The anthrax bacilli formed a distinct line of invasion in the living tissue and behind them the staphylococci were found in the non-staining material. The advanced necrosis in this position was evidently due to the secondary staphylococcic infection.

In another crypt, some distance from the carbuncle, a pure infection of anthrax bacilli was found. Surrounding this crypt the epithelium retained almost its normal structure, and yet large numbers of anthrax bacilli were seen between the cells penetrating into the surrounding inter-follicular tissue. The bacilli, while fairly frequent between the superficial cells, accumulated in enormous numbers in the deeper layers of the epithelium and in the subepithelial tissue. The crypt lumen contained quite a large number of free anthrax bacilli. Portions of the surrounding inter-follicular tissue showed congestion with beginning necrosis and there was considerable edema and extravisation of red blood cells into the diseased area. The cells in this area stained poorly and there was fragmentation of their nuclei and other evidence of karyorrhexis. The anthrax bacilli congregated around some of the neighboring lymphfollicles. While in some places they formed a net work around the capillaries, the bacilli themselves were not seen in the blood current. The surface epithelium, except directly over

the carbuncle, was intact and unaltered. In a few places along its surface a number of anthrax bacilli were caught and held in particles of mucus. The surface epithelium over the necrotic area had been destroyed and here large numbers of staphylococci were found, but no anthrax bacilli were seen in the epithelium itself.

Comment—It is evident from an examination of this specimen that the anthrax bacilli gained access to the parenchyma of the tonsil directly through the unaltered epithelium of the crypts. Having penetrated the superficial layers of the epithelium, they seemed to proliferate in the deeper layers before passing on into the interfollicular tissue. The toxines, elaborated by the anthrax bacilli, however, evidently influenced the vitality of the epithelium so that it was readily attacked by a secondary infection, and it is probable that the greater amount of destruction was due to the secondary invasion of the staphylococci. There was no evidence that the anthrax bacilli passed through the uninjured surface epithelium. The slowness in the progress of the disease in this case was due to low pathogenicity of the infecting organism, an attenuated strain of anthrax bacilli having been used.

Summary.

From a study of the experiments as outlined above, we find several important facts which warrant us at this time in drawing certain conclusions:

First.—The tonsils in the hog are more readily infected by the anthrax bacillus than any other portion of the buccal or pharyngeal mucosa. The clinical history of this disease in the hog shows that in a great majority of idiopathic cases, the pharynx is the site of the invasion, and in all of these cases of pharyngeal disease the tonsils are the site of the invasion, and in all of these cases of pharyngeal disease the tonsils are the portal of entrance. In none of my experiments was there any involvement of the pharyngeal or buccal mucosa other than the tonsils. While the culture of anthrax was generally brought into more intimate contact with one of the tonsils it was impossible to limit

the bacilli to the tonsillar surface and they must have come in contact with a large part of the pharynx. At the inoculation an effort was made to rub the emulsion into one tonsil only, and in only one case (Hog No. 5) were the lesions limited to only one tonsil, and in this case the tonsil affected was not the one on which the culture had been rubbed.

Second.—Anthrax bacillus penetrates through the cryptal and not the surface epithelium.

Third.—The Anthrax bacillus probably always gains access to the parenchyma of the tonsil by passing through the living, unaltered cryptal epithelium, and having gained access through the superficial layers of this epithelium they tend to multiply in the deeper layers and then pass into the inter-follicular tissue.

Fourth.—The anthrax bacillus penetrating through the living normal epithelium causes a devitalization of the tissue which paves the way for a secondary infection from the staphylococci or other pathogenic organisms.

Fifth.—The rapidity of the invasion is influenced both by the virulence of the organism used and by the susceptibility of the individual animal.

Following the invasion the subsequent course of the disease is similar to that found in other tissues. The toxin elaborated by the bacilli causes at first an accumulation of polymorphonuclear cells, later necrosis of the tissue cells with disintegration of the nuclei. The germinating follicles show more resistance to the disease than the inter-follicular tissue. Associated with the necrotic process is an increase in the number and engorgement of the capillaries, and sometimes there is marked extravasation of the red blood cells. The anthrax bacilli accumulate in the lymph spaces also around the blood vessel walls. In some of the sections examined the bacilli were found penetrating the blood vessel walls and a few were seen in the blood current. That these bacilli were not found in the heart blood was due to the fact that in anthrax the bacilli are held up in the capillaries until they have accumulated in numbers sufficient to overcome the vital resistance, when they are suddenly swept into the general blood stream.

Discussion.

Dr. Gordon Wilson, Chicago: One regrets that such an important paper should have a place at the end of our proceedings. It could only have been discussed satisfactorily had an opportunity been given for first seeing the microscopic sections. The anthrax bacillus certainly presents many advantages for such a research, but those of us who know anything about its virulence cannot but admire the fortitude of Dr. Wood, who, for the sake of these advantages, faces with equanimnity the dangers which working with such an organism entails.

The first thought that occurs to me is that it cannot be urged against the results that the abrasion caused by rubbing on of the bacillus was the centre and cause of the absorption, for in one case at least the affected tonsil was the one opposite to the focus of application. From what I have heard I find it difficult to acknowledge that the whole absorption was through the intact epithelium of the crypt.

Dr. Wood found the bacilli most abundant in the subepithelial tissue of the crypts and in the outer part of the follicles. Assuming, as Dr. Wood does, that they entered by the crypts, are we to believe that they progressed against the lymph stream? Might they not have reached the infected areas from adjacent territory, not necessarily from the crypts, and then multiplied? Are we to assume that any dilations made by Stohr's phenomenon play no part as a possible seat of entrance?

Dr. Wood, I gather, found no bacilli in the crypts nor in the center of the follicle. If they enter by the crypts, is one to assume that the crypts do not afford a good nidus for their growth? Also, we must assume that those which did not make good their entrance were speedily destroyed or washed away. This would be a very interesting observation. If they are going against the lymph stream, are we to assume that they never get so far as the center of a follicle, or getting so far, are speedily killed off? One thing is obvious, that once the organism gets entrance into the tonsil, it multiplies abundantly, and that despite this multiplication it may for long remain local.

Dr. George B. Wood, of Philadelphia: As far as not finding the anthrax bacilli in the main blood current when they were present in the capillaries is concerned, it must be remembered that in anthrax the bacilli accumulate in the capillaries in dense networks and do not become free in the blood current until

the last stages of the disease, when the resistance of the host has been entirely overcome. The danger of infection in experimenting with anthrax bacilli was not especially great, as they were always wet and could not become disseminated in the air. The cases of anthrax disease amongst those who handle skins are due to pulmonary infection by breathing in dried anthrax bacilli, especially the spores. An important further extension of this study would be in determining the resistance of the tonsillar tissues in the various breeds of hogs by the search for antibodies, bacteriolysin, agglutinins, etc., in a large number of tonsils taken from slaughtered animals. This has been suggested by the different degrees of infection caused in different animals by similar methods of inoculation. Practically the only fact demonstrated by the present research is that the anthrax bacilli will penetrate through the epithelium of the tonsillar crypt more readily than through the surface epithelium of the tonsil or other portions of the pharyngeal mucosa. The disease, however, having gained access to the tonsil, does not advance more readily opens up a large field for thought and experimentation.

PHLEGMONS OF THE UPPER RESPIRATORY TRACT.

Multa renascentur quae Tam coccidere.

BY PROF. FERD. MASSEI.

If I venture to offer this contribution (although modest) to so important a subject, it is because of the interest I have taken in it for many years, and of the fact that in spite of the rich literature many controversies exist.

These belong particularly to the classification of the different forms, and it must be borne in mind that the question is not simply a theoretical one, but one for many reasons very practical.

In the year 1885[1] I tried, with the support of fourteen personal observations, to demonstrate that several of the cases described as phlegmonous laryngitis were striking examples of primary erysipelas of the larynx. From Bouillaud and Ziemssen to Sir Morell Mackenzie and J. Solis-Cohen it was the first attempt to differentiate the so-called laryngeal oedema from phlegmon and from erysipelas, the latter being widely different, (1) on account of its general symptoms, (2) of its course, (3) of its constant point of departure (the base of the tongue and the glosso-epiglottic folds), (4) of the marked migratory character of the impairment, (5) of the easy diffusion to the lungs while the larynx remained quite free.

In the year 1888 Senator[2] called attention to a disease which, according to him, "was little known," to which he applied the name of "acute infectious phlegmon of the throat," marked by a diffuse and purulent inflammation of the deep tissues of the pharyngeal mucous membrane, which was apt to extend to the larynx and glands, and later to other organs. This opinion was strongly attacked by Guttman and Virchow, the first recognizing in the description an erysipela-

[1]Massei "Sulla erisipela della laringe," translated in German in the year 1886 (Ueber das primare Erysipel des Kehlkopfs.)

[2]See Berliner klin. Wochenschrift, No. 30, January.

tous inflammation; the second who suggested that it was not possible to fix exactly the relations between erysipelatous and diffuse phlegmonous affections.

In August of the year 1890, on the occasion of the X International Congress of Medicine held in Berlin, the topic, "Acute Infectious Phlegmon of the Pharynx and Larynx," was given to the late Professor Moritz Schmidt and to me.[3]

From my side I emphasized the possibility of a primary laryngeal erysipelas, and describing its characteristics, advanced the following conclusions:

1. In spite of the uncertainties of bacteriological researches, from a clinical standpoint, it is right to recognize that erysipelas may occur in the pharynx and in the larynx.

2. The attack may be primary; when the pharynx is involved, the larynx is always likewise affected; in case of a primary laryngeal erysipelas the pharynx may remain unaffected.

3. The disease may have an epidemic course.

4. Two forms may be recognized; one in which the local symptoms prevail, the other in which the general symptoms predominate.

My eminent colleague, Professor Schmidt, in extending his researches to the other form of phlegmon, as well as to those of the pharynx, recognized the difficulty of affirming the identity of the germs, and was favorable to making a distinction between them from the clinical standpoint, and discussed the differential characters.

Sir Felix Semon, in the year 1895, in the "Medico-Chirurgical Transactions," published an interesting pamphlet[4], in which after considering the chronological history of the phlegmonous affections of the throat, confessed that while his practical experience in the matter had not been large, and while he was not

[3]Verhandlungen des X Internationalen Medicinischen Congresses. Bd. IV., Berlin, 1872. Zwoelfte Abtheilung. Laryngologie und Rhinologie. See pages 173-187.

[4]Vol. 78. On the probable pathological identity of the various forms of acute septic inflammations of the throat and neck, hitherto described as acute oedema of the larynx, oedematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx and angina Ludovici.

able to present experimental proofs, nevertheless he believed that "acute oedema of the larynx, oedematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx, and angina Ludovici probably were identical pathological forms, from the point of view of their etiology, and that the severity and extension of the attack were in proportion to the degree of virulence and the eventual penetration of germs to various other parts of the body."

On the other hand, it is well known that there are several micro-organisms capable of producing acute septic inflammations of the throat, and that the Fehleisen's streptococcus is not other than the piogenus, whose virulence is simply increased.

After Semon's publication the enthusiasm with which my pamphlet on erysipelas of the larnyx had been accepted, in Germany particularly, lessened following publications (in the same Germany) accentuating the identity of germs apt to produce phlegmon, abscess and erysipelas, marked different classifications, and erysipelas was embodied in these various manifestations, losing its autonomy.

I do not intend to discuss the problem of the etiology; on the contrary, I prefer to accept fully that Fehleisen's streptococcus is not a specific one and I admit the identity of the germs of phlegmon and of erysipelas of the upper respiratory tract; but I beg to ask why, in spite of this, we continue to speak in general of phlegmon and erysipelas? Why we treat in a different manner a phlegmon and an erysipelas of the arm?

The distinction I have made for the throat is not referable to the cause; it concerns the clinical question, the first to which we must answer in our practie.

Clinically it has a great importance. While I was able to collect in a short time fourteen cases of primary erysipelas of the larynx, occurring in epidemic form, in a late period I have seen but rare cases and at long intervals of time.

If, then, the course, the symptoms, the phases, the prognosis and the treatment of the phlegmon, in whatsoever part of the body it develops, offer important differences with those of erysipelas, I do not understand why we refuse to admit this

distinction as regards the special regions of the throat and the larynx.

Erysipelas produces transient migratory lesions (except in the case of a special localization, as suppuration, phlictens, etc., in a circumscribed part of the tissue); phlegmon develops well localized and progressive changes, the one and the other sometimes dominated by general symptoms.

In consequence of such considerations, which I shall resume as shortly as possible, I believe that the following classification of the different forms produced by the same germs is preferable:

1. Above all distinguish the phlegmon of the upper respiratory tract from the oedematous form of laryngitis: either it is a question of a sero-ematic infiltration, or a purulent one, or a consequence of circulatory trouble, compression, ulceration (of different cause), or at last a simple angio-neurotic oedema.

2. Give a separate plate to erysipelas of the pharynx and larynx, whether primary or secondary to cutaneous erysipelas.

3. Recognize that primary erysipelas of the larynx commonly respects the pharynx.

4. Accord a separate place to the phlegmon of the pharynx and the larynx, or of both; two forms of which may be distinguished, the common and the septic (in which the local trouble is quickly overcome from the serious symptoms of adynamy and ipo-thermy, and which ends in a few days with asistolie;

5. And finally, the suprahyoidic phlegmon (or angina Ludovici), of which it is also useful to distinguish two forms: a benign one, apt to be treated (quick unbridle of the tissues), to which the larynx may or may not take part (collateral oedema, or even irruption of pus through the thyro-hyoid membrane); and another, a severe one (the septic), which is fatal often without suffocative symptoms.

The septic pharyngo-laryngeal phlegmon, it should be mentioned, has special notes: In some cases (which I observed and which happily are infrequent) death from syncope is the constant termination; the course is very short, adinamy very ac-

centuated; similarly striking is the foetor of the alitus; it may begin from the base of the tongue and the ary-epiglottic folds, but soon it reaches the tonsils and the faucial isthmus. Near the infiltration appear membranous patches, sometimes necrotic phlictens, which burst and discharge. Temperature, high at first, soon goes down to the normal. No Loeffler bacilli are found at the bacteriological examination; no treatment useful. If narrowing of the larynx is present and intubation is performed (which in such cases I prefer to tracheotomy), death happens through pronounced symptoms of general adinamy.

We know very little of the occasional causes; often it is spontaneous; once in my cases it was the consequence of scarlet fever. The late Professor Schmidt in his report has rightly recollected the possibility of foreign bodies, recognized by palpation and the laryngoscope.

The same can be said for the suprahyoid phlegmon; sometimes (in the septic form) the local symptoms before reaching a progressive course and showing the well-known symptoms and the tendency to asphyxia remain within circumscribed limits; characteristic hardness of the neck—and death without suffocative symptoms. Even a treatment in time does not prevent death.

Amongst the well-known causes I must mention caries of the teeth, or their extraction without the necessary aseptic precautions.

As it is a question of well-known fact I simply mention it, but it is sufficient to show that, from a practical standpoint, a distinction should be maintained between the morbid forms which, although having the same pathogenic cause, develop different manifestations which cannot be unrecognized and must be duly appreciated.

This conclusion in no way can modify Semon's observation, i. e., that there are cases in which even an anatomo-pathologist cannot say if the disease originated from the sub-cutaneous cellular tissue of the neck, in the tonsils or in the larynx, because I first admit these diffuse phlegmons, and accept their etiologi-

cal identity; but these are simply demonstrative examples which, as for their clinical signification, have nothing to do with erysipelas of the larynx, and particularly with the primary erysipelas, which has separate characters from those of the complex phenomenology of the phlegmon of the throat and, therefore, deserve to maintain its autonomy.

PERITONSILLAR PHLEGMON—REPORT OF A CASE.

BY F. E. HOPKINS, M.D.

Limited by the character of the case I have to report, my title should be phlegmon of the peritonsillar space, rather than the comprehensive one of "Phlegmon of the Upper Air Passages," under which it appears in the programme. The principal point which it illustrates is the possibility of erosion of large blood vessels.

As to the cause of peritonsillar infections we readily accept the bacteriologist's dictum that the throat at all times harbors bacteria ready marshalled for invasion, and since the crypts of lymphoid tissue are portals easily assailed, infections of this region should be frequent, and systemic involvement severe. The wonder is, not that phlegmons threatening life occasionally develop, but rather that such serious invasion is so rare an active professional life may pass and not a single case come under observation.

I leave to the bacteriologist the naming of the invading host and of the allies which join with eager savagery in the campaign; and I leave to him, too, the naming of the armies of defence both of the first and of the second line, adopting for myself the general terms of virulent or aggressive infection, and a lowered resistance on the part of the patient.

Pardon this seeming levity—it is only seeming. Bacteriological literature, with its explanations of infection and resistance, gives this kind of a picture, and though the language of this branch of science is not at my command, the reference will be understood.

Such microscopical examinations of pharyngeal phlegmon as I have on record give as a usual finding staphylococcus aureus, with in some cases a mixed infection—the most troublesome addition being streptococci. The staphylococci alone fulfill their contract for destruction with less systemic disturbance than when working in alliance with others.

However, microscopical findings are of little value in determining the treatment of these cases. The stage of progress decides that, and early, effective drainage is the best assurance of a favorable prognosis.

Peritonsillar phlegmon may run its course to serious complications with less systemic disturbance—as shown by prostration, temperature curve and pulse rate—than is the rule in follicular tonsillitis. The pain and distress may be great with the temperature not above 101 and a pulse of from 80 to 90.

Suffocation from flooding the larynx by the sudden rupture of the abscess has been reported, and tracheotomy has been required because of closure of the pharynx by infiltration and oedema; but the complication I wish to emphasize is that of erosion of blood vessels by the necrotic process.

When only the tonsillar artery is involved the hemorrhage may cease spontaneously or be easily controlled; if the ascending pharyngeal the situation is very serious, while a break in the wall of the internal carotid brings a swiftly fatal end. As illustrating invasion of the first or smallest of the three arteries, Dr. Bates, of Springfield, has recently described to me a case of active hemorrhage following the spontaneous rupture of peritonsillar abscess. Bleeding ceased without a resort to either pressure of ligation.

Bosworth's work, with its painstaking research of literature, was published twenty years ago, yet the chapter on peritonsillar phlegmon is still the fruitful source of reference. He gives a list of twenty authors, who, over a period of forty years, had reported cases of involvement of the large vessels during the course of phlegmon. Of all the cases reported but five recovered, and the inference is that in these only the smaller vessels were eroded.

Nearly all the recent text books speak of the possibility of this destructive invasion of blood vessels, but none of the authors I was able to consult mentions a case or refers to one.

The case whose brief history follows was under the care of Surgeon Major E. A. Gates, of Springfield, Mass. I saw the patient in consultation on May 23, 1900, and found: H. M., male, American, twenty-six years of age, of poor resistance

because of irregular life and recent acute illness. He had suffered from measles early in the month and while convalescing had taken cold—according to his own statement—the particular manifestation of this cold being a sore throat. There was a marked peritonsillar swelling upon the left side, with severe pain, but the patient was not prostrated, being at this time dressed and about the house. His pulse was 80 and temperature 100; and during most of this illness the pulse rose only to 80 or 90 and the temperature at no time exceeded 101. No fluctuation could be made out. I saw him again on the 24th, and although the cellular infiltration had increased, there was no evidence of "pointing," nor could fluctuation be felt. Deep incisions were made, but these yielded no pus and little bleeding took place. Two days later a hemorrhage occurred which the attending physician controlled by pressure. A terrific hemorrhage recurred on the 27th and proved quickly fatal. A post mortem was not allowed, but such examination as could be made immediately following death showed rupture through the posterior pillar—the flood from the eroded carotid finding exit here.

Such a mass of cellular infiltration should be explored by a blunt instrument, even the finger, following unfruitful incision. In this case the patient—an only and spoiled child—objected vigorously, as did the parents; and as a peritonsillar abscess usually soon ruptures into the incision or burrows its way to the surface, further interference was not insisted upon, with the unusual result named.

PHLEGMONS OF THE AIR PASSAGES—INFLAMMATION OF THE LATERAL COLUMNS OF THE PHARYNX LEADING TO ABSCESS FORMATION, WITH REPORT OF CASES.

BY HENRY L. SWAIN, M.D.

When a tissue like the lymphoid in the naso-pharynx and throat, possessed of certain peculiar characteristics, becomes inflamed in any one of its various portions, it might reasonably be expected that certain results would follow. Or, to put it in another way, if certain kinds of inflammation are possible in any one part of Waldeyer's ring, it certainly ought logically to be possible in any other portion. Any isolated mass of lymphoid tissue could, therefore, in a general way, be expected to act, when inflamed, exactly after the fashion of the faucial tonsil, and could have, like the latter, simple acute, follicular, rheumatic, diphtheritic and phlegmonous inflammations. As knowledge became more widespread concerning what happened to the adenoid or pharyngeal tonsil we learned to know as years went by that it might have acute as well as chronic inflammation. The same was true of the lingual tonsil, and at a meeting of our association in '96 at Pittsburg it was my pleasure and privilege to read a paper chronicling various cases of acute inflammation of the lingual tonsil which ended in abscess formation.

In recent years much has been written in the journals concerning inflammation of the lateral columns of the pharynx, and I have a presentiment that if we continue to remove, root and branch, from our young children all of their adenoids and faucial tonsils there will be abundant need for us to devote increasingly more and more attention to these masses of lymphoid tissue as well as to the lingual tonsil. Hundreds of observations exist to attest to the fact that when, in years gone by, adenoids were removed in young children and the faucial

tonsils were left untouched the latter subsequently enlarged into a perfectly healthy growth as though needed by the system, and as though trying to supply something to satisfy this lack or want. If they, the faucials, are thus nowadays also ruthlessly removed there will, just as surely as the sun rises and sets, be enlargement of the lingual tonsil or lateral columns of the pharynx chronicled, as there has been of the faucials following the older simple adenoid operation. And if this is logical and true, we should find not only acute inflammation of these structures, but also the phlegmonous.

What was true way back in '96, before the war upon the adenoid tissue was as general and as destructive as in recent years, is, of course, as you all can bear me out, perfectly true of the present time. Not only have you and I been seeing cases of quinsy of the lingual tonsil from time to time, but it has been my fortune to see several within the last few months. In one of the last cases there was a great similarity noted, which was suggested in my earlier paper, to Ludwig's angina. There was here, curiously enough, enormous swelling of the sub-maxillary glands, inability to shut the mouth owing to the swelling in the floor of the mouth, and elevation and protrusion of the tongue. It was impossible to get the mouth open far enough to admit a finger, still less to make an examination with the mirror, and through no skill of mine the diagnosis was finally made, after the worst danger was over, when the swelling subsided owing to rupture of the abscess into the throat. Examination of the lingual tonsil, when possible, revealed the spot of rupture at one side of the center. What was of even more importance in a way, as illustrating the danger the patient ran, was the observation of a subsiding swelling of the epiglottis and ary-epiglottic fold which went way round to and included the arytenoids. The patient had therefore been in extremely threatening danger by reason of the oedema of the glottis and could have readily died from suffocation, as has been frequently observed to be the case in real Ludwig's angina.

This latter possibility actually occurred only a week previously in the case of one of my ablest colleagues, who was

operating to open the abscess of a genuine Ludwig's angina. He was compelled to make a brilliant emergency tracheotomy, rescuing the patient from immediate death. When respiration was restored he proceeded to find the abscess and drained it splendidly, only to lose the patient some days afterward by sudden hemorrhage, of unknown origin, into the windpipe.

These two cases were immediately followed in my own experience by the last case that I have observed of abscess of the lateral column of the pharynx, the kind of phlegmon which I wish particularly to emphasize in this communication.

We have assumed more or less arbitrarily that as the lateral column of the pharynx is no mean mass of lymphoid tissue, and as it has been known to have all of the other kinds of inflammation to which lymphoid tissue is heir, we have a logical right to expect that there might also be the phlegmonous type, and I propose to submit the facts concerning certain cases I have observed to prove that such may be the case. If this is, however, as logical as I have indicated, it would seem strange that no one would have thought it worth while mentioning, unless, as I shrewdly suspect, few have believed it really to occur. Even so patient and persistent a writer and observer as Levinstein, of Berlin, who has with great detail told about all kinds of inflammation in the anginas of the lateral columns, as he calls them, fails to say anything definite about such a quinsy.* I have not found it mentioned anywhere. Indeed, few of the textbooks think it is even necessary to mention the lateral column of the pharynx, probably reasoning that it would behave as any other lymphoid tissue did, and what was the use of talking about it? Moure, of Bordeaux, in his book comes as near to what I am considering as anybody that I have happened to discover. He is very thorough in his pictures and in his descriptions of the anatomical relations of the faucial tonsil, and says that he finds a great many times the abscesses of the faucial tonsil point posteriorly.

Take this body of gentlemen present and listening to these

*The same is true of Henke in the last number of Fraenkel's Archivs (Bd. 27), who writes under the title of "Phlegmons of the Palate Tonsils."

words of mine, representing the best thought and broadest erudition of any group of men in the country in this particular line of work which we have before us, and if abscess of the lateral columns of the pharynx was of frequent occurrence it would seem as though we must have all seen it many times. I am sure you will all, if you think a moment, recall cases of quinsy where you had tried to puncture and failed, which burst in the night, and, nothing showing in front, you concluded it must have perforated posteriorly. Indeed, in some instances you have been able to absolutely observe the pus coming down from behind the palate. I myself have had numerous occasions where I have been unable to get at the abscess and have contented myself with the idea that the pus had not yet formed, only to later be forced to acknowledge that it was there all the time, and that in following the routine plan of making incisions I did not get to the spot where the pus was. It is easy enough if you have a pocket of pus and it shows white under the epithelial coating to know enough to cut in there and evacuate it. And that was just how I stumbled upon my first case of abscess of the lateral column of the pharynx. And then others followed, and I now have six or seven "sure enough" cases, my last one being within a week, as I have said, in a lad of thirteen.

As the subjective and local symptoms of all of these cases were about alike, it will not be necessary now to detain you but a very few minutes to describe what happened in one case which is doubly interesting in the fact that, like quinsies of the faucial tonsils, these of the lateral column seem to have the tendency to recur. All of my cases have been in young people. All of them have had edema (except this last case of the boy of 13), and the edema has, in the case of the recurrent quinsy of the lateral column, been of the most threatening type.

In the case of a young student in our university whose history I am about to relate there seems to be no doubt that we have had three genuine occurrences of abscess in the same situation, and I am responsible for the fact that two of them were in the lateral column of the pharynx, in the same region and on the same side.

The patient, a young man of nineteen, had been in my charge, at the Yale Infirmary, for two weeks, with an active double otitis media. One side, the left, had subsided quickly; the other, being much more active, gave us much more trouble to control. At the end of the second week he suddenly had a very sharp rise of temperature and a chill. He broke out with the most profuse erythematous eruption that I have ever seen. The body was completely covered with an urticaria, as it proved to be, and the first thought was that we had to do with a very active case of scarlet fever. With the beginning of the hives he had a very marked case of indigestion, if one chooses to use the term. The patient was nauseated and vomited; could retain nothing on his stomach for forty-eight hours. In the middle of this period he began to have a sore throat. This latter symptom was, owing to the various other things which had to be done for him, somewhat neglected. It had been going on in a mild degree for two days before it assumed so active proportions that we must needs pay attention to it. It was on the left side, the side on which the ear, fortunately, seemed to be entirely through with its acute stage. The tonsil of that side swelled and began to bulge out into the throat, being forced forward and downward toward the median line in quite the typical way for quinsy, if one did not observe carefully the situation. Being somewhat hoarse on the third day of his apparent quinsy, we discovered that he had a very marked edema of the aryepiglottic fold of that side. The arytenoid of that side was swollen as large as your thumb, a very large, baggy swelling. At this time of the discovery of the edema we attempted to find where the abscess was, making two endeavors before the fact was revealed that the tonsil was not swollen in the way which it usually is, or at least not displaced in the way in which it usually is, and that there was considerable space between the palate and the posterior pharyngeal wall. Poking forward the posterior pillar, I discovered that the lateral column of the pharynx was enormously swollen, and with a great effort (for, as is usual, the mouth opened with great difficulty and one could see in only through a very small aperture) we hooked the palate forward sufficiently to discover that the upper part

of the column of the pharynx was very much swollen and pushed toward the palate. Taking my cue from two previous cases of abscess in this region, which I had formerly had, I made the incision around up in back of the palate into the swollen area and evacuated a large amount of pus. At this time it was learned from his parents, who had come upon the scene, that he had on a previous occasion been troubled with a sore throat, where he had the utmost difficulty one night in breathing, and they feared that he would suffocate, and where relief had come by the rupture of the abscess. No observations had been made as to where the abscess formed, but from my description of the situation as it was then, the mother felt very sure that the conditions had been exactly the same in the previous attack. In any case, they had not been able to see the place where the pus came from, following its evacuation. With the subsidence of the swelling and the retreat of the edema the urticaria vanished and the patient rapidly got well. During these days of observation which immediately followed the abscess I was able to absolutely ascertain that the opening was in the substance of the lateral column.

A year and a half later I was called to the infirmary to see this same boy, reporting that he had another attack of quinsy, which had not gone on so long untreated as before, in which there was again just the beginning of edematous swelling about the arytenoid of that side. But knowing from the previous history just where to go to get at the pus-pocket. within twelve hours of the onset of the severe symptoms we located and opened the abscess, and, as nearly as I can tell you, it was in precisely the same spot.

It is interesting to chronicle that the hives were also present in a most profuse way and that the patient suffered with an upset digestive tract.

Commenting on this case, many interesting points occur to us. That the skin could have, together with the other tissues, what amounted almost to an edema would seem to indicate that possibly the edema of the larynx in this case was also constitutional or systemic in origin, and this leads us to state that the whole matter of edema of the larynx would seem to need a

fresh rehearsal. It would be interesting to have us all, in the next few years, observe carefully our cases of edema and to see if we could establish some theory of their existence which is less open to objection than the old venous stasis theory, which has generally been held, when one had a foot, or a hand, or a leg, and he desired to make clear why the swelling took place.

The investigations of Fisher, of Cleveland, have led him to believe that the edema is, in a sense, a local acidosis, and the cells of the capillaries allow of the escape of fluid into the tissues, which in a state of health they do not permit. No theory of pressure or damming back of the circulation could explain, to my mind at least, why a region like the aryepiglottic fold, which receives blood from an entirely different set of vessels from those which supply the tonsil, and whose lymphatics evacuate very much lower into those of the neck, could, by any pressure or stasis which the abscess in the tonsil itself could make, be made edematous when so far away and so out of all immediate contact with the active purulent process. Hence one could in this case quite readily accept Fisher's idea that there has been brought about in some way this local acidosis, although it is still difficult to understand why the local acidosis should be found in the aryepiglottic fold and not in some other portion of the body, assuming as we do that there is no immediate direct connection in the circulation, venous or lymphatic, between the two areas.

Now, I have recently, in this season, had in my own experience six cases of edema of the larynx. In two of them there was no quinsy at all. There was inflammation of the lateral column of the pharynx in both these instances, and there was in one a most marked general, what may be called rheumatic infection, where various joints of the body were swollen and inflamed, with rapid jumps from these to other joints, and with no persistence of the symptoms in any one place for any length of time. In three other cases in other years mumps seemed to be the cause of the edema of the laryngeal region, which removes the cause of pressure or stasis or infection still farther away from the larynx than in any case of quinsy or inflammation of the faucial tonsil or lateral column. So that from these

various cases of edema one has to look to some other cause than pressure, and the general acidosis of the system which seemed to have taken place in this case of lateral column phlegmon on these two occasions seems to afford a reasonable explanation of why he could have edema in any place in the body. If the system had been so disturbed as to produce this rise of temperature, erythematous urticaria, and upset digestion, it could also make the swelling in the region of the larynx. We have still to explain why it occurred first upon the side of the inflammation in the lateral column and secondly on the other side to a slighter degree.

Examination of the urine in this case showed high acidity, large indican reaction and no albumin or sugar.

The well-known curious vagaries of the so-called angio-neurotic edema point all too clearly to the fact that local areas of edema can take place in almost any part of the body, even where there is no systemic or kidney condition to be looked upon as the causal agent.

The second interesting thing which we might dwell upon for a moment in this case is the fact of the recurrence on the same side, in essentially the same region, just exactly as we have so frequently observed in quinsies of the faucial tonsil.

The third point is one which any one desiring to argue could well maintain—was this not after all an abscess of the tonsil which simply pointed posteriorly, and in the process of so doing caused infiltration and swelling of the lateral column of the pharynx? The only answer that I have to make to this is that I did see here the swelling and abscess so intimately in the substance of the lateral column that I do not believe it was a quinsy of the faucial tonsil. My reason for asserting that the more flatly is the fact that in these six cases of so-called abscess of the lateral column the swelling or the displacement of the tonsil has been really different from that usually observed in true quinsy cases. To my mind in the true quinsy case there is almost always swelling of the tonsil itself, as well as displacement of it forwards and downwards toward the median line. In these marked cases of abscess of the lateral column the pillars of the palate, the tonsil and the whole soft palate are all

displaced, but there is almost no swelling of the tonsil itself, and the posterior pillar of the palate is pushed forward and away from the pharynx wall; whereas when abscess occurs in the dead space above and laterally from the faucial tonsil, the usual peritonsillar abscess, there is almost always a pressure backward of the posterior pillar of the palate, bringing it into more immediate contact with the posterior pharyngeal wall than would be the case with the opposite side of the throat. So that, if I have rightly observed the situation, I am almost sure that there is a pronounced difference in the swelling in the two instances which one can readily observe if he carefully watches the position of the posterior pillar of the palate.

Of the other cases that I have to chronicle, one was a young lady of twenty, who had never had a quinsy to her knowledge before, which was the first case that I had observed in whom there was also edema of the glottis, and where I made three distinct attempts to discover the abscess by puncturing into the peritonsillar space in the usual situation, only to discover that I had not been observing the case properly and that the abscess formation was really back of the palate. Here it was absolutely forced upon me by seeing the abscess practically protruding around the edge of the palate when the patient gagged, and the evacuation of the abscess was followed by the usual quick relief.

Moure speaks also of abscesses occurring in the substance of the posterior pillar of the palate. This I have also seen, and it is to my mind and eye a very different situation from what I am trying to describe as existing in the substance of the lateral column.

Between the two attacks in the patient first mentioned I saw another student with the mumps who developed a supposed quinsy, but the real abscess formation was in the lateral column of the pharynx as in these other instances, and he had a very profound edema which was most threatening in its aspects.

The fifth case was in a young man of twenty-two, with nothing particular to distinguish it from the others, the symptoms being all the same as in the case of the young lady first ob-

served. It was a second abscess, the previous one having been in the usual situation back of the tonsil of the opposite side.

The last case, which I have so recently seen, was in the lad of thirteen, who had never had a previous known attack of quinsy and where the lateral column was enormously swollen at the start. His tonsils had been removed, were very small and only a little swollen during this attack. The first puncture of the lateral column brought out no pus, and I thought I had made a mistake in my diagnosis, and on the second occasion tried to find pus in the usual region, but failed also here. In the night the abscess ruptured, and the point of evacuation was in the lateral column, out of sight of direct vision, behind the palate.

There is nothing so particularly urgent in this presentment that should necessarily claim your attention for so long a time in a meeting of this kind only the general interest which we all take in peculiar and rare phenomena, and the hope that I have that in having mentioned this form of abscess not only will some case be clearer to those of you who are here, but that perhaps the profession in general may by observing a little closer make their earlier incisions more effective and thus, as sometimes in my own cases I know, may save the patient from twelve to twenty-four hours of suffering, not to mention the relief to the pressure which in some way is causing an edema of the larynx. And a word here: Whereas all these cases of oedema have transpired without any actual serious interference with breathing or the symptoms being what might actually be called dangerous to life, yet I cannot contemplate the existence of a large edematous swelling on one or both sides of the larynx without feeling that the patient is seriously in danger of suffocation, and, as every other thing, one hears of cases that have ended fatally because of profound swelling, the possibility of locating the abscess a little earlier is one which may perhaps be a life-saving device. In any case, I am grateful to you for the attention that has been accorded the paper.

PHLEGMONS OF THE UPPER RESPIRATORY TRACT.

BY JOHN O. ROE, M.D.

The word "phlegmon" (from the Greek φλεγω to burn) was originally used to denote a flame, fire, heat or inflammation. It may be mentioned as a curious fact that it is derived from the same root as "phlegm," which was regarded by the ancients as one of the four humors which composed the human body. But the word "phlegmon" came to be restricted in medical science to denote some form of inflammation, which meaning it has retained until the present day.

Phlegmon, therefore, is simply another name for inflammation, although clinically it is regarded as of an extraordinary intense inflammation and looked upon as being of a more or less malignant character. Thus, we may have an inflammation of this character in any part of the body, the intensity of which is dependent upon the virulence of the infection and the resistance, or lack of resistance, of the patient, or of the tissues, to such infection. The use of the word phlegmonous inflammation, therefore, may be regarded as a superlative expression, being used to indicate clinically that the inflammation is of an unusually severe form or grade. Thus, if in the case of a tonsillitis the inflammation is more active and more intense than usual, involving the deeper cellular structures of the neck; if in the pharynx, the inflammation extends to the deeper retro-pharyngeal spaces, terminating perhaps in a retro-pharyngeal abscess; if involving the nasal passages and accessory sinus, the inflammation rapidly extends to the mininges and, if instead of the ordinary simple laryngitis the inflammation extends to the peri-laryngeal and tracheal spaces, and, if suffocation does not speedily ensue, terminating in mediastidnal abscess—in such cases as these the inflammation is regarded as phlegmonous. Inflammations of this severe grade termed "phlegmon" may therefore involve any or every portion of the upper respiratory

tract, conditioned only, as I have stated, by the virulence of the infection.

We almost invariably find that persons attacked by phlegmon are those whose general condition has become greatly weakened and whose defenses have been broken down by some other diseased condition or dyscrasia, such as diabetes, chronic nephritis, a prolonged illness, like intermittent or typhoid fever; the debilitating influence of extreme or prolonged fatigue or the auto-intoxication from the chemotoxines that infect the system and insidiously undermine the systemic forces, rendering the patient vulnerable to all sorts of microbian attacks. As is readily seen, it is out of the question to consider specifically these multifarious conditions, and the only practical addition to our knowledge of these conditions is by reporting cases that come under our observation.

Case 1 is that of a phlegmon of the fauces, terminating in retro-pharyngeal and a retro-oesophageal abscess and death. A young man, twenty years of age, a student in the university, had been out to a late class supper and ate very heartily, being followed almost immediately by the symptoms of a hard cold. The following day he was ill with diarrhoea, headache and severe sore throat. He grew rapidly worse, his neck became much swollen, particularly on the right side. He was hoarse and spat blood from what was thought to be an abscess of the tonsil that had opened spontaneously. I saw him on the fourth day, when I found the whole fauces intensely inflamed and swollen, and the pharynx on the right side bulging forward, presenting to the finger the doughy feel of the retro-pharyngeal abscess. This I at once opened, giving vent to much bloody and purulent matter. The track of the abscess began quite rapidly to extend downward until it became retro-oespohageal. The discharge of pus was very copious, at times so profuse as to nearly suffocate him, his breath seeming to pass through a quantity of soap suds. Temperature 103 and pulse 120. Several physicians saw him with me, but in spite of all that could be done he continued to grow worse, the bloody and purulent discharge increased, respiration became more difficult, he became rapidly weaker and died from exhaustion on the third

day after I saw him. No post mortem, I regret to say, was permitted. The infection was a mixed infection, streptococci predominating.

Case 2 was that of a young woman, eighteen years of age, who had what was thought to be diphtheria. Two days before she had been taken with a severe chill and at the time I saw her her temperature was 103. The fauces were enormously swollen and there was a profuse thick, tenacious exudate covering the fauces, but not a membranous formation. The bacteriological examination showed it to be a mixed infection, the pneumococcus and streptococcus predominated. The inflammatory action extended downward, involving the laryngeal and pharyngeal tissues. The dyspnoea became so urgent that tracheotomy became necessary, but notwithstanding every effort to save her, she sank and died five days after the onset of the disease. Previous to her illness she had become very much run down from overwork at school and preparations for her examinations.

In several cases of faucial phlegmon occurring in children from three to twelve years old the inflammation resulted in post-pharyngeal abscess. In all these cases the abscess was opened and the patient made a rapid recovery.

Through the kindness of my friend, Dr Thomas Hubbard, of Toledo, I am able to report three cases that have come under his observation representing three different types. First, the phlegmonous angina, followed by retro-pharyngeal abscess. Second, angina with laryngo-tracheal oedema and metastases. Third, scarlitinal phlegmonous angina. His report of these cases is as follows:

Case 1.—A middle-aged physician with clean health record had a severe pharyngitis with fever, chills, etc., and by the third day it had developed a very severe phlegmon, involving the hypo-pharynx, larynx and probably the oesophagus. The swelling quite prevented the swallowing of even liquids for several days. Dyspnoea and local pain made sleep impossible. The neck, especially the right side, was swollen and stiff; in fact, it was an unusually serious type of angina. The temperature was 104 maximum; albumen and casts in urine. The

treatment was chiefly local—steam inhalation, impregnated with carbolic acid in alkaline solution, was used almost constantly and moist heat externally. The dysphagia made general medication per oris impreaticable; in fact, this was about all that could be done. The carbolized steam seemed to check the progressive oedema and lessen the pain, and in about forty-eight hours the swelling in the hypo-pharynx subsided and deglutition was resumed. On close inquiry, I verified the statement that the patient was usually in extraordinary good health; had not a bad habit, and, aside from the fact that his vocation brought him into contact with all classes of acute infections, there were no other pre-disposing causes. The subsequent history is interesting. The acute celluitis subsided, and in less than a week he exhibited only a moderate degree of glandular infiltration on the right side. This condition persisted and dysphagia returned. He journeyed forty miles to enter a hospital, when a more thorough study was made. Daily temperature of 101 degrees F., albumen and casts in urine, throat culture early in illness showed streptococci. Blood cultures on entering the hospital negative. The hypo-pharyngeal swelling became more pronounced, and in about a week it was apparent that pus was forming. An incision was made as low down as possible in the posterior pharyngeal wall and nearly an ounce of fetid pus was evacuated. This was swabbed out daily with dilute iodine solution and gradually healed. Convalescence was slow, and months afterward he had not recovered his former standard of health. His splendid constitution had overcome this severe infection, but not without evidence of impairment of vitality.

Case 2.—Man, aged about fifty, hitherto healthy, had on Friday, January 26. 1912, a sudden onset of throat symptoms, so severe that he was compelled to leave work. He did not seem to have had much fever, but just a gradually progressive oedema of hypo-pharynx and larynx, and when I saw him three days later he had been in a state of extreme dyspnoea for several hours. He was hurried to the hospital and tracheotomy under local anaesthesia was done. The tracheal mucosa was oedematous, but there was comparatively little general celluli-

tis of the neck tissue. He rallied promptly and seemed perfectly comfortable. No febrile reaction, no systemic disturbance, and as soon as he could swallow he took abundant nourishment. The voice became natural in two days. On the second day the tracheal wound was much inflamed and infiltrated, and gradually there devoleped evidence of septic phlebitis spreading downward into the thorax. The tracheal cannula was removed on the third day and there was no recurrence of oedema of larynx or hypo-pharynx. A metastastic abscess formed in the right leg and on the fifth day this was opened. This pus contained staphylococci only. The patient died of general sepsis on the sixth day. Streptococcic serum was tried in frequent large doses prior to the abscess findings, but without appreciable effect.

Case 3.—A young woman contracted scarlet fever from her children and during the second week developed a very severe phlegmonous angina. The tonsils and peritonsillar tissue were much swollen, deglutition was quite impossible, and there was considerable dyspnoea. Sepsis was in evidence, rapid pulse and low temperature, and the throat was covered with a thin, gray membrane. There was a constant discharge of muco-pus from naso-pharynx and nose. Systemic condition fairly good, no naseau, and normal urinary secretion. When first seen in consulation I was suspicious of pus formation in peritonsillar tissue and made free incision well into the supra tonsillar fossa. No pus, but considerable relief of tension pains. Carbolized steam inhalation was given every hour and serum treatment was begun as soon as fresh serum could be obtained from the husband's blood; that is, in twelve hours. The sepsis increased, manifested by somnolence, delirium and increase of general cervical swelling, with only a moderate degree of fever, 103 degrees F. The prognosis was very grave—it seemed that natural resistance was totally exhausted. This patient had been Mayoed a few years previous for exophthalmic goitre and was a poor subject for such a virulent infection. Cultures showed pure streptococci. She responded to the second injection of husband's serum—that is, the faucial swelling began to subside about twelve hours after the second dose,

10cc. In all, 80cc. were administered at intervals of six hours, and the convincing feature of the therapeutic value of the serum was the rapid convalescence. I have never seen scarlatinal angina clear up so quickly.

Through the kindness of Dr. Sargent F. Snow, of Syracuse, I am able to report an interesting case that came under his observation last year. A lady, aged fifty-eight, began to have a sore throat and to feel badly on June 30. On the following day Dr. Snow saw her in consultation with the family physician, who had regarded it as a case of quinsy. Dr. Snow found a general firm infiltration of the whole left side of pharynx, but no particular point of prominence, although the tonsil was covered with a dirty yellowish deposit, not membrane. This was washed off and the tonsils scarified freely with deep incision, both anterior and posterior, with the hope of relief from the local depletion, although no pus was found. Hot saline irrigations of the throat were ordered, with free calomel purgation. In spite of all this there was a rapid increase of the infiltration and a general phlegmonous condition, so that on the following day the breathing was very difficult. The urine had become loaded with albumin, with evidence of an extreme general mixed infection. Upon consultation it was decided that further operative measures would be useless, and the patient died the following day.

A peculiar circumstance attending this case was that two days previous to her being taken ill she was in Dr. Snow's office with her sister, who was under his care. The doctor was much impressed by her general appearance at that time and urged her to see her family physician, for he feared from her color and apparent condition that she was a very sick woman. She laughed at the doctor and said "she never felt better," but within twenty-four hours the conditions developed, as described above. "The case as a whole," said Dr. Snow, "was one of the most impressive evidences of the intimate relation between general active auto-intoxication and acute inflammatory condition that I had ever seen."

Fatal hemorrhages may also result from such phlegmons, as

in a case reported by J. D. Rolleston[1], as follows: A girl, aged six, was admitted to the hospital on October 22, certified to be suffering from diphtheria. Excepting an attack of whooping-cough at three years, she had until recently been a healthy child and there was no family history of bleeding. Five weeks before admission she had measles, since when her voice had been husky. On October 20 she complained of sore throat, and on the 22d she had a croopy cough and the neck glands became swollen.

Condition on admission—Deposit on both tonsils and uvula, slight nasal discharge, voice husky, stridor, croupy cough and dysphagia. Temperature 100 degrees F.; 16,000 units of antitoxin were given. October 23, deposit clearing away, leaving marked ulceration of tonsils and uvula. No fetor. No Vincent's organism found in the throat smear; temperature 103 to 100 degrees F.

October 24, dysphagia less. Temperature 102 4-10 degrees F. to 99 degrees F.

October 25, at 9:00 a. m., she suddenly gave a cough and a huge gush of blood poured out of her mouth. Death, preceded by marked cyanosis, occurred within five minutes. No diphtheria bacilli, but only cocci, were found in three successive cultures from the throat and one from the nose.

Necropsy, October 26—abscess cavities in both tonsils, ulceration of the uvula, soft palate, epiglottis, fraenum-epiglottidis, valleculae and ary-piglottidean folds. Deep ulceration of laryngeal portion of pharynx, exposing muscular tissue. Three small superficial ulcers above right vocal cord. The exact site of the bleeding vessel was not determined, but there was no evidence of erosion of external carotid, internal carotid or internal jugular, nor of any glandular abscess. There was no history or evidence of scarlet fever, no fetor or other evidence of gangrene, no clinical and bacteriological features of Vincent's angina. It is highly probable, the author thinks, that the pharynx and larynx were predisposed to infection by the recent attack of measles, especially as the child's voice was still husky when the fatal illness began. This form of sore

[1]"British Journal of Children's Diseases, Feb., 1913."

throat, the author says, is usually due to streptococci, often accompanied by laryngeal involvement, especially when it is secondary to measles, when it frequently runs a severe course. The author also cites thirty-five cases recently reported by Goodall[1], twenty-five of which were fatal. In nine of these cases the larynx was involved, although the principal lesions were in the pharynx, and two had recently had measles.

In sporadic cases phlegmons nearly always assume a virulent form. The onset of the affection is sudden and its progress very rapid, and in adults the majority of cases prove fatal in from thirty-six to seventy-two hours. Sir Felix Semon described several such cases in a lecture on "Acute Septic Inflammation of the Throat and Neck[2]," delivered at the Long Island College Hospital November 3, 1904.

The disease is usually ushered in with a chill and with pharyngeal symptoms, attended with dysphagia. The tissues of the throat are oedematous and the glands of the neck usually become swollen—sometimes enormously so. The patient is hoarse, which quickly becomes complete aphonia, soon followed by dyspnoea, often necessitating tracheotomy. In some cases the inflammation is confined to one side of the throat. Semon, in his lecture already referred to, reports a case of inflammation of the larynx which was unilateral. He also classifies all these different septic inflammations of the throat and neck that have been known by different names, because manifesting different symptoms when different parts are involved, under the one comprehensive term of "Acute Septic Inflammation of the Throat and Neck," including even the well-known disease called Angina Ludovici, named after the good old Doctor Ludwig, of Strassburg, who in 1830 first described the particular manifestations attending septic inflammation of the sub-maxillary gland and the surrounding tissues. As several cases of this disease happened to come under his observation, with manifestations entirely different from any cases he had seen before, he thought he had discovered a new disease.

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[1]"On a Fatal Variety of Ulcerative Sore Throat," "Metrop. Asyl. Board's Ann. Rep. Med. Supp.," New York, 1909, p. 245.

[2]Brooklyn Medical Journal, Nov., 1905.

The bacteriological findings in these cases are usually a mixed infection, although streptococci is the one germ that predominates. It is also found to be most virulent in the complications that arise in connection with these phlegmons, such as pericarditis, nephritis, pneumonia, pleurisy and peritonitis, which therefore supervene because of its virulence, while other general septic conditions may be due to the different strains of the streptococci.

In the epidemic form, which sometimes prevails in different sections, the disease is less fatal than in the sporadic cases. The virulent infection in this form is evidently fostered by atmospheric conditions, as in epidemics termed "La Grippe," or transported sometimes by water pollution, and sometimes by the milk supply. Several outbreaks of tonsillitis of a peculiar nature, such as the English term, "Septic Sore Throat," have occurred in this country, the source of which in most cases was traceable quite directly to an infected milk supply. Such an outbreak occurred in Boston, Brookline and Cambridge, and was traced by Winslow[1] to the portion of the milk supply of the Deerfoot Farm Dairy that came from the Southboro District. He was able to get fairly complete reports of about 1,000 cases which were observed in the vicinity of Boston, and 400 in Hudson, Marlboro and Southboro, with forty-eight deaths in all, attributed directly to the disease. His description of the disease is as follows:

"The throat sometimes showed white patches characteristic of ordinary tonsillitis, and in other cases a membrane was formed like that in diphtheria, while others were marked only by a diffused redness. The most striking feature, however, was a secondary enlargement of the glands of the neck, which in many cases followed on the first sharp throat attack and which in some instances was followed by a general invasion of the deeper tissues, leading to sepsis, rheumatism, erysipelas, nephritis and other maladies."

Winslow also says that he was not aware that this form of

[1]"An Outbreak of Tonsillitis or Septic Sore Throat in Eastern Massachusetts and Its Relation to an Infected Milk Supply." Journal of Infectious Diseases, 1912, v. 10, p. 73.

throat disease had before been noted in this country. A precisely similar outbreak, however, has occurred several times in Great Britain[1], and in each case was traced to milk. A similar outbreak of septic sore throat of a mild character, mainly among children, occurred in the city of Schenectady last year. Sixty-three cases were reported by Van der Bogert,[2] of which only six were over fifteen years of age. The majority, twenty-six, were between the ages of one and five. Thirteen occurred in infants under one year, and thirteen between the ages of ten and fifteen. The majority of the cases are reported as tonsillitis. In twenty-eight the cervical glands were enlarged, in eighteen the sub-maxillary, in nine the parotid, in eight the anterior cervical, in ten the sub-mastoid, and in four the posterior cervical; the others were the lingual, sub-lingual and the left cervical. There were pains in the joints and rheumatism in three; four developed nephritis; one pneumonia, and four were complicated with bronchitis; two had quinsy; two had earache, and four developed otitis. There were no fatalities in any of the cases. The source of the milk supply as the suspected cause of the epidemic was investigated, and it was found that fifty-three of the patients were supplied by thirty-four distributors, and as there were but two cases at the most occurring in one milkman's route, the committee were led to believe that the milk supply should be absolutely disregarded as a positive factor.

During the winter of 1911-1912 epidemics[3] of septic sore

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[1]For a record of "Similar Outbreaks in Other Countries," vide Winslow's article, just referred to.

[2]Archives of Pediatrics, May, 1912.

[3]"Epidemic of Sore Throat Due to Milk: A Preliminary Report," by J. L. Miller and J. A. Capps, Jour. Am. Med. Assn., Chicago, 1912, Vol. LVIII., p. 1111. Also "The Chicago Epidemic of Streptococcus Sore Throat and Its Relation to the Milk Supply," by Joseph A. Capps and Joseph L. Miller, Jour. Am. Med. Assn., Chicago, 1912, Vol. LVIII., P. 1848.

throat appeared both in Chicago and in Baltimore[1], traceable directly to an infected milk supply.

The epidemics in Boston and Chicago were largely confined to adults, whereas in Baltimore, as in Schenectady, they mainly prevailed among the children. In studying the bacteriology of these epidemics they were found to be due almost exclusively to a variety of streptococcus. In Baltimore it was estimated that there were not less than 1,000 persons infected during the outbreak and possibly more, with 48 deaths, while in Chicago it was believed that there were not less than 10,000 persons affected by the epidemic. The particulars regarding the death rate in this epidemic are fragmentary and indefinite. There are 622 cases reported as having been investigated with a record of 19 fatal cases.

In the Baltimore epidemic, in addition to the streptococci, Hirshberg[2] found pneumococci in 48 cases, while in the Chicago epidemic Davis and Rosenow[3] isolated a peculiar streptococcus, which gave rise to a large number of complications and many deaths. The disease was characterized by a sudden onset of severe prostration, glandular enlargement and frequently by other serious complications. The organism isolated was an encapsulated hemolytic streptococcus of high virulence, which always acidifies and sometimes coagulates milk.

It is interesting to note that in the suspected herds cases of mastitis were found and in some instances unsuspected cows were discharging pus with their milk.

In studying the relationship of this peculiar streptococcus,

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[1]"An Epidemic of Septic Sore Throat in Baltimore and Its Relation to a Milk Supply: A Preliminary Report," by Louis R. Hamburger, Jour. Am. Med. Assn., Chicago, 1912, Vol. LVIII., p. 1109.
"The Baltimore Epidemic of Streptococcus or Septic Sore Throat and Its Relation to a Milk Supply," by Louis P. Hamburger, Vol. XXIV., p. 1-12. (Bull. of the Johns Hopkins Hosp., Baltimore, 1913.)
Also "Septic Sore Throat—A Milk-Borne Outbreak in Baltimore, Md.," by Wade H. Frost, Passed Asst. Surgeon U. S. Pub. Health Service. Pub. Health Rep., Vol. XXVII., p. 1889. "Bacteriological Study of the Outbreak," by Wm. Royal Stokes, M.D., and F. W. Hatchell, Pub. Health Rep. Vol. XXVII., p. 1923.

[2]"Malignant Pneumococcus Tonsillitis." Journal American Medical Association, Chicago, 1912, Vol. LVIII., p. 1189.

[3]"An Epidemic of Sore Throat Due to a Peculiar Streptococcus." Journal American Medical Association, 1912. Vol. LVIII., p. 773.

isolated by Davis and Rosenow, Hersberger regards it as related to the streptococcus pyogenes group. These groups Leutscher[1] classifies as follows:

1.—The usual streptococcus pyogenes group, which does not clot milk or ferment raffinose.

2.—The streptococci angiosus group, which usually clots milk and often ferments raffinose.

The streptococcus pyogenous group is usually associated with suppurations, erysipelas and septicemias.

The streptococcus angiosous group is especially associated with inflamed throats, scarlatinal and otherwise. It is also found in malignant endocarditis, peritonitis and otitis.

While in these epidemics, reported as a "milk-borne outbreak" and attributed to the streptococci because of their great predominance, it cannot be said in any case that the disease has been caused by any one organism.

So is it also in the sporadic cases, for in none can the phlegmon be attributed to any one particular germ or organism, however largely the streptococcus may predominate. Semon found in all his cases a mixed infection, so that in none of the cases could he attribute the intensity of the phlegmon to any one particular organism. Neither can the source of an organism be determined by an examination or culture of the germ. In studying the streptococcus occurring in milk, and also in the throat, Stowell, Hilliard and Seglesinger[2] in their conclusions said:

"Two hundred and forty pure strains of streptococcus have been compared as to their morphology, gramstrain reaction character, and amount of growth and their quantitative acid production in seven carbohydrate and related organic substances. We have not found that any of the features, other than the fermentative reactions, are sufficiently correlated with one another or with acid production to assist in fixing the organisms in groups or in distinguishing the source of isolation."

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[1]Bacteriology of Epidemic Sore Throat, Baltimore, by John R. Leutscher, Jour. Am. Assn., 1912. Vol. LVIX., p. 869.

[2]A statistical study of the streptococci from milk and from the human throat. Journal of Infectious Diseases, 1913, Vol. XII., p. 144.

From a study of these phlegmons in their various relations the conclusion must be reached that the virulence of the disease depends more largely upon the condition of the patient—that is the soil in which the germ becomes implanted—than upon the strain or character of the germ, the activity of the germ depending upon the condition that quickens it into activity, which may be local or general, or both combined. Chronic affection of the throat may provide in many cases the inviting condition for the activities of wandering germs. Farmers and fanciers recognize the importance of introducing new strains in order to increase the vigor and productiveness of their animals; so may it be that the introduction of a new strain of streptococci into the old saphrophytes starts a new and prodigious activity. Davis[1] in a bacteriological study of chronically diseased tonsils and their relation to arthritis, nephritis and endocarditis almost invariably found in the crypts large quantities of virulent hemolitic streptococci fatal to guinea pigs and rabbits, with joint and other manifestations. These germs were ever ready to take on an active form of existence when excited to activity by external influences or given a chance by the weakening of the defenses of their host. While the patient may sometimes claim that he is feeling "exceedingly well," or "never felt better in his life," he may be at the same time in a condition to afford a savory morsel to some voracious germ. In a private communication from my friend, Dr. Jonathan Wright, regarding this subject, he says:

"We have known for years that streptococci are carried as saprophytes in the normal throat totally innoxious to the bearer, but sometimes deadly to his neighbor. They must depend for their virulence upon systemic variations of the host. When external conditions have an influence upon the host which induces this specific change in the host's throat tissues we have a hitherto innoxious inhabitant of the throat become a deadly enemy.

This way of looking upon throat infections, not only of the

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[1]"Bacteriology and pathology of the tonsils with special reference to chronic articular, renal and cardiac lesions." Journal Infectious Diseases, 1912, Vol. X., p. 148.

septic variety but in tuberculosis and in other infectious diseases, I have long entertained. While milk streptococci may be capable of inducing such epidemics when these unknown external conditions render the throat ripe for it, it behooves us primal cause of these epidemics."

Discussion.

Dr. Greenfield Sluder, of St. Louis, Mo.: It seems to me that there is one feature which has not been clearly brought out in the papers—the seriousness of peritonsillar or phlegmonous sore throats. The cases reported by Dr. Casselberry prove the identity of these sore throats with erysipelas, and I would like to add this testimony: Last Winter I was called to see a man with a violent sore throat, without any special localization. There was a high grade of edema of the soft palate which extended downward to the arytenoid cartilages, where it seemed to stop. There was sloughing of the tissue of the soft palate. The next day the tip of his nose became reddened, and it looked like a case of erysipelas. This spread over the body, proved to be erysipelas, and resulted in killing the patient on the second day. There was a streptococcus infection behind without admixture apparently.

REPORT OF A CASE OF PHLEGMON STARTING AS A PERITONSILLAR ABSCESS AND EXTENDING DOWNWARD AS FAR AS THE SECOND RING OF THE TRACHEA.

BY GEO. L. RICHARDS, M.D.

The following case is an example of a severe case of pharyngeal phlegmon, having its origin primarily in a peritonsillar abscess and secondarily in a diseased tonsil. The patient was a large, well-nourished man, forty-two years of age, of English parentage. The family history was negative. The previous history was that he had suffered from occasional quinsy sore throat for the past three years, having had two or three attacks. Otherwise he had been well. The patient presented himself in my service at the Union Hospital on the afternoon of August 20, 1909, with a history of a sore throat of several days' duration, followed by difficulty in breathing and swelling of the entire left side of the face and neck. The left tonsil had been lanced once the previous day and again on the morning of admission, but without relief, and the abscess had been continually increasing in size. At the time he presented himself he was breathing with great difficulty, and with occasional paroxysms of dyspnea and coughing. The entire left side of the face and neck clear down to the collarbone was swollen and edematous. On examination of the throat, which was made with some difficulty, the tonsil and lateral wall and the supratonsillar area were intensely swollen, and largely filled the available area in the throat. The patient was nearly cyanotic, with a pulse of 124. The skin of the neck was very tense, but I did not feel any definite fluctuation. I assumed that I had to do with a large peritonsillar abscess, and sent him at once to the operating room. He was seated in a chair, a knife gotten ready, the tongue depressed, and an incision made in the supratonsillar fossa. This brought pus, and also a paroxysm of coughing. As the first opening had been more in

the nature of a diagnosis as to the site of the pus and was only a small one, the patient was quieted and then an attempt made to enlarge the opening. With the first enlargement of the opening there was a gush of pus, at the same moment an inspiratory spasm, a quick cry and cessation of breathing. He was immediately placed on an operating table, but by this time respiration had ceased. Unfortunately, nothing was ready for a tracheotomy, and in the sudden shifting from the chair to the table the knife had been dropped on the floor and momentarily lost. A hurry call for a knife brought me first a thin nasal chisel, which the interne in his hurry had mistaken for a knife, and a delay of several seconds occurred before a knife was obtained. The head was placed somewhat over the edge of the table, the neck on the stretch, and a quick incision made at the supposed site of the first and second rings. Much to my surprise, I entered not the trachea but an abscess cavity and found that the abscess from above had burrowed downward, producing a phlegmonous inflammation of the tissues of the neck, and had pushed the entire trachea and thyroid area well over toward the right, and itself extended down to a little below the second ring of the trachea. The lower abscess being opened, and while still flowing pus, I was able with the finger to orient the trachea, which was then quickly opened. The patient by this time was apparently lifeless, but the pulse was still beating. Artificial respiration brought inspiration after a few seconds and in a short time breathing was re-established and a tracheotomy tube inserted. The abscess cavity in the tonsil was then widely opened with the aid of the finger as well as the abscess cavity at the site of the trachea. The two were found to communicate. The patient was placed in bed with the feet about a foot higher than the head, and kept in this position for the next three or four days, a wick being inserted around the tracheotomy tube. The after history was uneventful, the temperature, which was 101½ on admission, slowly going down. There was an abundant pus discharge from both the mouth and at the side of the tracheotomy tube for the first few days. The tracheotomy tube was removed on the eighth day and he was discharged from the

hospital cured on the seventeenth day, the neck at this time being nearly healed. The drainage tube was continued until complete closure of the skin wound. He was advised to have the tonsils removed, but never came back to have it done. I have recently looked up the case and find that he has been perfectly well for the last three years. This case illustrates the possibility of danger in a case of peritonsillar abscess and the absolute necessity of the evacuation of such an abscess; also the danger in allowing such an abscess to go on to the point where it discharges itself. It is sometimes stated that such an abscess will eventually discharge itself just about as well as when assisted. This does not seem to me to be the case. While it is sometimes true that they will find exit from the peritonsillar fossa at the junction of the anterior and posterior pillars it is never safe to wait for this. While cases such as I have described occur relatively seldom, the possibility of the extension of a purulent process down the neck must be borne in mind. The course of the pus probably corresponds to the course of the bucco pharyngeal fascia, which invests the constrictor muscles of the pharynx, and is given off from the prevertebral fascia, this latter fascia forming its posterior limit. The superior constrictor muscle, which is in close contact with the tonsil is in relation externally with the prevertebral fascia. In this case the pus probably forced its way through the fibres of the superior constrictor muscle, thus gaining access to the prevertebral fascia, thence following down the neck along the line of this fascia, thus coming in contact with the larynx and trachea, since this fascia forms the posterior limit of a fibrous compartment which contains the larynx, trachea, thyroid gland, pharynx and oesophagus.

An error which I made here should also be avoided, namely, the attempt to open any such extensive phlegmonous collection of pus without being prepared for a tracheotomy. Again, so extensive an abscess should have been opened in the first place with the individual on the back. Pressure on the pneumograstric probably accounted for the severe dyspnea. As to exactly why the inspiration of the pus should have caused cessation of respiration rather than a paroxysm of coughing I am

unable to state, unless relief of pressure on the pneumogastric produced for the moment some temporary paralysis or disturbance in function. I believe it is true that sometimes sudden cessation of pressure may produce some such effect on the respiratory centre.

Discussion.

Dr. Henry L. Wagner, of San Francisco; I believe that pathologists in their research work on phlegmons are inclined to have a different view than heretofore; there are various conditions which produce phlegmons, and sooner or later a change in the classification will take place. It is known that a streptococcus infection does not always produce conditions of this kind. It is very important, as already pointed out, that systemic disturbances should be considered foremost.

I will report a most peculiar case of a descending phlegmonous inflammation produced by the pneumococcus; it was not a streptococcus infection. It began in the eye, and then travelled downward into the pharynx and larynx.

The etiology of the oedema has been ably spoken of by Dr. Swain. Oedema was caused by an altered condition of the blood vessels. In the nasal cavities it was observed in some cases of anaphylaxis.

Dr. William E. Casselberry, of Chicago: For many years these phlegmonous inflammations have been looked upon as erysipelas of the throat, presumably a secondary infection by whatever strain of streptococcus might be the cause of facial erysipelas. This view, at first, was based upon the occurrence of severe throat attacks either in conjunction with or following erysipelas of the skin and, latterly, upon the observation that facial erysipelas itself commonly originates in and spreads from the nares, especially so in connection with accessory nasal sinus suppuration. But in a case recently observed by me a secondary erysipelas of the face and body followed a direct autoinoculation from a primary phlegmon of the throat through the medium of fingernail scratching. During the recent epidemic of streptococcus pharyngitis, a middle-aged woman was suffering from severe throat symptoms which culminated quickly in obstruction to breathing. The palate was very oedematous and leathery and the velum, dragging on a swollen tongue, was the cause of the dyspnoea as the larynx was not oedematous. Though the appearance was not typical of peritonsillar abscess, punctures were made in order to exclude it, no pus being then found, but the escape of serum relieved the ten-

sion and obviated the need of tracheotomy. After a day or so, the extreme symptoms having subsided, some pus was seen to be escaping from one of the punctures. She had a habit of moistening her finger in the mouth and of then forcing it well into the canal of the ear to relieve itching. After five or six days on her complaint of the ear, examination showed a moist oedematous condition of the external auditory canal, which by the next day was obviously an erysipelas which rapidly extended over the auricle and then over the whole face and part of the body, running a course of about fourteen days, with recovery. The throat cultures were reported by the Chicago Laboratory as showing pneumococci and streptococci, but without other details. The oedematous leathery thickening of the mucous tissue observed in this case is a characteristic of the erypsipelatous type of phlegmon of the throat and consists largely of a submucous cellulitis comparable to the cellulitis of cutaneous erysipelas. Should the larynx be involved suffocation is apt to culminate rapidly and I had three other cases in which unconsciousness had already resulted before I was called to open the air passage. After the operation each of these patients revived for a few hours, but, as usual, if prolonged suction by violent inspiratory effort be not surgically obviated short of the moribund condition, all three of them succumbed to secondary oedema of the lungs. Dr. Richards is to be congratulated on having secured a permanent recovery under such circumstances.

But not all so-called phlegmons of the throat are of the erysipelatous type, as simple circumscribed abscess, so situated as to compress or involve the larynx may cause similar symptoms and justify the term phlegmonous, as, in still another case in which infection through the lingual tonsil, secondary to nasal sinus suppuration, had culminated in a deep abscess under the base of the tongue. It was reached and the pus evacuated by an angular laryngeal lancet after penetrating to a depth of 2 cm., going through the swollen lingual tonsil and then with considerable force through seemingly a dense muscular fascia. The resistance of this fascia had been felt and the necessity of penetrating it realized in consequence of failure to do so at two immediately preceding punctures. The incision was made under illumination by a laryngeal mirror. The hemorrhage, though disquieting, yielded after a time to digital compression. Relief was immediate and permanent.

Dr. D. Crosby Greene, Jr., of Boston: I believe that in many cases where the inflammation is limited to one side of the throat, the oedema of one side of the larynx being definitely marked with external swelling of that side, median incision

is the method of choice for obtaining drainage. This has been impressed upon me especially by two cases that I have seen recently.

I recently had a case in which this condition obtained. There was an acute infection, streptococcic in nature, with oedema of one side of the larynx and a definite external swelling on that side. The oedema was increasing and accompanied by difficult respiration and it seemed advisable to make either an internal or external incision for drainage. I made an incision in the median line from the upper border of the thyroid above, to a little below the cricoid cartilage, and dissected the soft tissues off the thyroid in the direction of the swelling. Before reaching the posterior border of the thyroid there was a gush of sanguinous fluid; this was followed by relief from the pain and dyspnea almost immediately. The operation was done under a local anaesthetic. One advantage in making this incision was that the exposure of the trachea at the lower end of the wound made instant tracheotomy if necessary a very simple procedure. This patient recovered rapidly from his local infection, but in three days developed a streptococcic pneumonia, from which he also recovered. I have known of several other cases that have been incised and drained successfully by median incision. This is a resource which should always be borne in mind.

Dr. Cornelius G. Coakley, of New York: The cases related represent two classes clinically. In one class, if we can get a good light and look down, we will see a definite area of fluctuation which can be opened through the mouth, but this is not the kind of cases that require our serious consideration. The cases I find that terminate so frequently fatally are those with a very moderate or slight tonsilitis; they recover from the tonsilitis and then complain of an exceedingly sore throat and difficulty in swallowing, and nothing can be seen at first on examining the throat. Such cases frequently occur in short necked, fat individuals, and they are hard to examine. Sometimes we find nothing for twenty-four (24) hours; then there will be seen or felt a hard swelling, beginning edema on the side of the wall of the pharynx and in from twenty-four (24) to forty-eight (48) hours the patient would die of suffocation. If tracheotomy is performed the patient usually dies of a septic pneumonia. If an internal cut is made, one will often fail to find pus. The edema will increase and the patient die. I have tried the internal operation, but I feel that the only way to relieve these patients is by the external operation. The incision is made along the sterno-cleidomastoid muscle. Many of these patients have died of an acute sup-

purative edema, such as follows the presence of a foreign body. In two cases that died I found only little blood and serum in the swollen region in the neck. These are the most serious types of cases. An external incision made early is the correct one.

Dr. James E. Logan, of Kansas City: I believe that the classification made by Dr. Coakley is a very important one. In many of these cases it is very hard to find a distinct fluctuation or any evidence of pus.

I saw an interesting case in February. The patient for a day or two had a slight tonsillitis, from which he recovered. About three or four days after he was taken suddenly ill with an edematous swelling of the left palatal fold extending down over the epiglottis, and a distinct edematous mass could be seen on the left arytenoid cartilage. It struck me that this was a very serious condition.

I would suggest a treatment, that of the application of ice. First give these patients a purgative of calomel with castor oil following. Then make these cold applications for sixty (60) hours or longer, until the edematous condition disappears. This has been my experience. In the case I reported I think this sore throat was a causative factor. This patient the evening before had partaken of a hearty dinner and I believe that conditions of the alimentary canal should be taken more into consideration in the development of these edematous conditions. There is also a close relationship with angioneurotic edema. I am opposed to scarification in these instances and think the application of ice results in great benefit.

Dr. Robert C. Myles, of New York: The subject has had my serious consideration during the last few years, for I have had some very desperate cases to deal with in my practice. The proposition as to whether an internal or an external incision should be made should be discussed and analyzed. I have seen but one death from a phlegmon of the upper respiratory tract. Recovery in these cases, I believe, is due to surgical interference probably aided by injections of antitoxins. We use ice bags over the part. We often see cases that are so desperate that they alarm their families even. I often have wished that some one would take up and report on autopsies. I am often puzzled *where* to cut in many of these cases. In certain phlegmons there would be but a small focus of pus and yet the condition would be found to be most serious; this one focus must be reached before relief can be had.

So far as the edema is concerned, I believe that it is due to the physical laws of osmosis and I remember one case, the relation of which I think will be of interest.

I was called at 6 a. m. to see a woman forty (40) years old, who was moribund. She had edema of the ventricle and over the cord and she had just enough breathing to sustain her. I made an incision posterior to the tonsil. The swelling was of such extent that it went above the hyoid fossa. I entered a tube-like fossa two inches long and in five minutes she obtained complete relief. The condition, I believe, was due to pressure upon the superior thyroid veins, either direct or indirect. Another incision was made low down and a string placed in. The wound healed and the patient has been well ever since.

Last winter I saw a woman, seventy-three (73) years old, who had an abscess beneath the tonsil and which extended to the hyoid fossa. This was split open. I had a diffieult time keeping her from strangling. There was present an edema, evidently a suction edema.

I advocate the external operation when other methods fail to give relief.

Dr. John R. Winslow, of Baltimore: One year ago this month I saw a case that was similar to the one reported by Dr. Richards. This was a young colored man who had been suffering from an attack of "septic tonsillitis," a disease then prevalent. This was probably followed by a peritonsillar abscess. When he appeared at the clinic six weeks later he complained of soreness on the side of the neck and difficulty in swallowing, but he was not suffering from dyspnoea. There was a marked edema of the perilaryngeal tissues on the left side and of the epiglottis, but no swelling or obstruction inside of the larynx. A fine thread of pus trickled over the left side of the epiglottis, but the source could not be determined. When the patient entered the hosiptal it was thought an external operation was called for. During the night, however, under hot applications, the abscess burst and the patient left the hospital and disappeared from observation for six weeks. He then returned with the history that in the interim there had been five or six spontaneous ruptures of abscesses. He was very emaciated, dyspnoeic; there was marked subglottic stenosis and he was in such a bad condition that it was determined to do a tracheotomy. One and a half hours before the time appointed for doing this operation I received a telephone message that the patient was dying, and after hastening to the hospital an emergency operation was done under local anaesthesia. The minute I incised the soft tissues there gushed out a large volume of pus. The tracheal wall was enormously thickened. After opening the trachea the patient collapsed and was apparently dead. We used oxygen gas and amylnitrite delivered into the

trachea through the wound. Eventually he recovered from this and was in a very fair condition. The patient was kept under observation for many weeks thereafter. Ultimately he got in good general health, but he has never been able to do without his tracheal tube. The larynx seemed to be in a condition of extreme collapse. Either there was a destruction of the cartilage, converting the laryngeal box into a bag, or there was a paralysis of all of the laryngeal muscles. This case I believe to be one of a neglected quinsy with gravitation downward in the tissues of the neck and development of a perilaryngeal and peritracheal phlegmon.

Dr. Greenfield Sluder, of St. Louis: It seems to me that there is one feature that has not been clearly brought out in the papers, the seriousness of peritonsillar or phlegmonous sore throats. Aside from the cases narrated this feature adds to the importance of such tragic affairs. The cases reported by Dr. Casselberry prove the identity of these sore throats with erysipelas, and I would like to add this testimony: Last Winter I was called to see a man with a violent sore throat without any special localization. There was a high grade of edema of the soft palate which extended downward to the arytenoid cartilages, where it seemed to stop. There was sloughing of the tissues to a certain extent. The next day the tissues became reddened and it looked like a case of erysipelas. This spread over the body, proved to be erysipelas, and resulted in killing the patient on the second day. There was a streptococcus infection behind without admixture apparently.

Dr. Emil Mayer, of New York: This discussion calls to mind an address delivered in Brooklyn by Sir Felix Semon on septic infections; attention was called to the fact that these infections appeared so quickly that they caused death in a very short time. It seemed like the story "their patients died or they recovered." But how about the patients that recover temporarily with the tracheal tube in position? I have had a case where tracheotomy had to be performed in a hurry; the patient was found to be choked up when the tube was removed, and it had to be replaced in a hurry; this was necessitated because of an infolding of a fold of the epiglottis. The epiglottis fell in and choked the patient. I removed the epiglottis and the patient has been perfectly well since.

Dr. Henry L. Swain, of New Haven: I will refer to a case occurring in my town which is similar to the one Dr. Richards reported. The patient was a physician. The abscess had pointed toward the middle of the neck at the sternal end of the clavicle on one side, and subsequently on the other. There was so great a stiffness of the muscles of the neck that it was

nearly a year before he could raise his chin into proper position.

With regard to the external incision in Ludwig's angina, I have mentioned in my paper a case in which it became necessary to do a tracheotomy where there was employed an instrument similar to that of Dr. J. Solis Cohen for bloodless tracheotomy, a very handy thing. The incision was made along the sterno-cleido-mastoid muscle. The patient died a few days later from sudden and profuse hemorrhage, and practically drowned in his own blood.

I do not believe that we paid enough attention to such a thing as the possibility of abscess of the lingual tonsil when dealing with these cases of phlegmon, which appear to be extending down the neck.

Dr. John O. Roe, of Rochester: In closing the discussion I would say that any inflammation of the upper air passages, or of any part, whether due to diphtheria, erysipelas, scarlet fever, measles or what not, is a phlegmon. This is a term that is used very loosely, for we often see such inflammation when unusually intense referred to as a phlegmonous inflammation, which simply means an inflammatory inflammation and which is, as I have stated, simply a tautology.

With regard to the constitutional conditions associated with these septic inflammations, and which play so important a part in the causation of the diseased process, it must be observed that this condition is very frequently overlooked, for it is the preceding toxic condition of the patient that provides such an excellent breeding ground for pathologic germs with which the patient may become infected, and it is by reason of the fact that the patient's defences have been broken down by this preseptic condition that these acute inflammatory processes are so frequently fatal. It is, therefore, in the treatment of these cases of the utmost importance to take into consideration such constitutional conditions. Local treatment also is of importance, as the use of ice, and such measures as have been spoken of by Dr. Logan and Dr. Myles.

Dr. George L. Richards, of Fall River: I have had one death occurring from edema of the larynx. I was called out at night, ten (10) miles from home, to see a woman who had no pain, no temperature, but who did have edema of the soft palate and arches adjacent. She was breathing with difficulty. I made incisions into the edematous tissues and the difficult breathing was relieved. The patient was then left in charge of the family physician. The next morning I was informed that six hours after she had suddenly stopped breathing. I thought that probably I should have done a tracheotomy, as an emergency operation in this case.

FURTHER OBSERVATIONS ON SOME ANATOMICAL AND CLINICAL RELATIONS OF THE SPHENOID SINUS TO THE CAVERNOUS SINUS AND THE THIRD, FOURTH, FIFTH, SIXTH AND VIDIAN NERVES.

BY GREENFIELD SLUDER, M.D.

Last May it was my privilege to present this subject to this Association in the shape of a preliminary report. Now I should like to add a note to it. The subject is a large one and clinically deals with a most difficult class of cases. It may be recalled that the question of the aetiology and treatment of migrain was raised in that report; and the statement made that I believed many (but not all) of the recurrent headaches that bear the name migrain are sphenoidal empyemata that have lost most, if not all, local signs; or were started as such empyemata; and that the nerve trunks have become involved either by extension of the inflammation (or its toxin) through the thin wall separating the sphenoid sinus from the adjacent nerve trunks. The results (case reports) that I might now submit, although a goodly number, are too recent to permit of conclusions. I feel that these results should stand five years before conclusions are drawn. As far as they go, however, they strengthen my belief in the correctness of the deductions detailed in that report.

I desire now especially to record an anatomical observation and to mention a conclusion drawn from clinical observation as far as it has gone.

Last May I cited that the treatises on anatomy usually gave the impression that the trunks of the 3d, 4th, 5th, 6th and Vidian nerves were well (far) separated from the sphenoid sinus. During the year an additional number of volumes have been consulted, but no exception to this rule has been found. The observation that the 3d, 4th and the three divisions of the 5th, 6th and Vidian frequently lie in close association to the

sphenoid sinus was a deduction from specimens which I had studied by cross sections. Two months later (July 10, 1912) the Archiv für Laryngologie, B. XXVI. (II. Heft) was published, including an anatomical investigation by Ladislaus Onodi, of this same district, with corresponding findings, save for the Vidian nerve, which he did not observe. His method was to follow the nerve trunks in certain specimens, sometimes to remove the wall of the sphenoid sinus and then to study the relations of the nerve trunks thereby exposed. He found that they were in these close associations for varying distances, sometimes even as much as 20 m.m. He did not consider the cavernous sinus in these relationships. He pictures specimens, however, where the sphenoid sinus extended to close approach to the clivus of Blumenbach—so close as to make transparency of the separating bone—and shows how this brings the 6th into these associations. I have not such a specimen. Five years ago, while looking over the exhibit by Dr. Mosher in the Harvard Medical School, I saw a number of such; but none of sufficiently thin wall to suggest their being classed with such material as I drew my conclusions from. It is interesting to know the length of such contacts; but I believe this to be of secondary importance to the fact that the exposure or contact exists, however short or long. Last February I enjoyed the personal favor of an inspection with Dr. Warren B. Davis, Keen Research Fellow of the Jefferson Medical College, Philadelphia, of his matchless collection of 145 Caucasian specimens showing the nose and accessory sinuses from the eighth week of foetal life to the twenty-fifth year uninterrupted, several specimens for each year (save the eleventh year—only one).* With his sanction I now record the observation made then, that the sphenoid sinus spreads laterally at an early age, reaching to close proximity to the 2d division of the 5th as early as two and one-half years; and that this condition runs almost constant throughout the series. Its development (Davis) begins on the anterolateral aspect of the body and slowly extends

*These specimens were secured by the ingenious technique which Dr. Davis was clever enough to devise; which he will publish in a monograph giving his observations on the collection.

backward. It, however, spreads rapidly laterally to approach the foramen rotundum, and then proceeds backward. As early as the sixth year the Vidian canal may be approached.

These observations seem, to me, of great importance. They furnish anatomical findings to correspond with the clinical histories of this class of cases: i. e., that the neuralgias frequently begin in quite early life. For if I be right in the conclusion that the mode of production of these headaches—the pathological sequence—is the close association of the sphenoid sinus to these nerve trunks and that the inflammatory processes are transmitted through the thin bone separating the cavity of the sphenoid from the associated nerve trunks, it is necessary that such anatomical associations be formed in early life as an explanation of such headaches beginning in early life.

Last May I made the suggestion that I thought the pathological process underlying these cases to be a hyperplastic sphenoiditis. A year more of clinical observation strengthens this belief, although some cases are certainly not such, according to their clinical behavior; and bone removed from some of these patients was declared to be normal by Dr. E. L. Opie.

From an observation of about one hundred cases it has seemed that the 2d division of the 5th and the Vidian are the nerves most frequently involved (95%). They may be involved singly; or together, then making the picture which would otherwise emanate from the sphenopalatine ganglion.* The year's observation leads me to feel it to be more difficult that I once thought to separate this class of cases from such neuralgia, so that I feel that one ought to be carefully on guard for this differentiation.

The third nerve is rather frequently involved; but I cannot now give percentages. Such patients are usually unconscious of a difference in their pupils, but upon observation show this difference with almost every coryza. Observations relative to the 4th nerve are not so readily made. So far I have not seen a case of 6th nerve involvement belonging in this category.

*The Syndrome of Sphenopalatine Ganglion Neurosis, by Greenfield Sluder. Am. Jour. Med. Sciences, Dec., 1910. Trans. Am. Laryng. Assn., 1910.

During the year I have tried for intrasphenoidal use three medicines not mentioned in the first text. Iodide of potassium in water 2%-5% proved to possess nothing upon which its use may be recommended. It is apparently inert. 1% Chlorotone in water proved to lack recommendations. The proprietary "cresatin" also failed to prove better adapted for these purposes than carbolic acid or oil of wintergreen.

The medicines which have so far given best satisfaction are 1% carbolic acid in oil, 2%-10% oil of wintergreen, and aqueous solutions of sodium salicylate, 2%-5%. These have been successful in allaying the pain, which persisted long after the sinus was satisfactorily opened and healed.

Discussion.

Dr. William Henry Haskin, of New York City, by invitation: I am particularly interested in the anatomical portion of the paper and what has been presented is very similar to what I have discovered.

I agree with what was said regarding the frequency of the second division of the fifth and the Vidian, which belonged to it, being in contact with the sphenoidal sinus. I have many specimens which show the second division of the fifth nerve in contact and several showing the third branch of the fifth also in contact. With regard to the third, fourth, ophthalmic division of the fifth and sixth nerves, out of two hundred and fifty specimens I have yet to see if they are in contact in the sphenoidal sinus. The carotid artery, with its sympathetic plexus, passes through the carotid canal and then takes a distinct turn upwards and passes forward along the sphenoid in the cavernous sinus. After passing forward to the spheno-maxillary fissure, it turns upward and backward. The above nerves pass over the carotid artery as it enters the cavernous sinus and are on the outer wall of the sinus throughout.

The one thing Dr. Sluder fails to grasp might explain most of the symptoms in the spheno-palatine region and other conditions, and also the large number of ocular manifestations that occur in nasal sinuistic and aural diseases. The sympathetic nerve passes along the inner side of the cavernous sinus and forms the cavernous plexus. This supplies sympathetic fibers to the ciliary ganglion, as well as sympathetic fibers to the spheno-palatine which join the Vidian nerve.

In studying the physiology of the vaso-motor supply that is the sympathetic nerve, we find that every one of the symptoms described in the papers—I have carefully read over about two hundred and fifty of them—can readily be accounted for. There was not present any neuritis or choked disk: what had been described were not true cases of neuritis or choked disk; these pointed to some intracranial pressure or actual meningitis.

I am interested in the trophic nerves, i. e., the sympathetic nerves which control nutrition. In any sympathetic nerve disturbance in connection with the fibers that pass to the optic, there occurs a variation in the blood supply to the optic nerve. If there happens to be any destruction of the sympathetic nerve about the sphenoidal sinus it is very plausible to suppose there will result actual trophic disturbances of the optic nerve and one will get quite a true picture of optic atrophy. The trophic supply is lost because of this disturbance of the sympathetic nerve. The sympathetic nerve is in direct contact with an area of bone which is liable to inflammation. It seems that most all the manifestations can be explained if we study the sympathetic nerve supply more carefully. At a meeting last May Dr. Sluder spoke of relieving the pain in glaucoma by cocainizing Meckel's ganglion. In reality he relieved the pressure through the sympathetic; every manifestation in the eyes has been described as the result of some irritation of the sympathetic nerve fibers and has been proven by experimental work by the physiologists.

Paralysis of the sixth nerve is more often met with than believed. This nerve pierces the dura mater in the inferior petrosal sinus and then enters the cavernous sinus, and is liable to become involved in any sinus thrombosis.

Dr. Sluder: In closing, I would say I am very much interested in Dr. Haskin's discussion and the points that he raised. However, most of them are irrelevant to the subject he has just presented. Dr. Haskin spoke of various troubles with the eyes, as well as headache, all of which he explains by the wide distribution of the sympathetic nerve. This idea, however, has been brought forward before by Dr. J. Schnetter, of New York City, in 1889, who brings out the idea that all kinds of headaches result from the cavernous plexuses about the cavernous sinus being in such close relationship to the sphenoidal sinus. The subject matter of my paper seems to me to raise an entirely different question.

Concerning glaucoma, which was mentioned by Dr. Haskin, I have not had the opportunity of observing very many cases —about twenty-six in all. In all of these cases, however, the

pain of the glaucoma has been uniformly controlled by cocainizing or injecting the sphenopalatine ganglion. I consider it a nerve blocking process. Sometimes a simple cocainization seems curative, at least for the painful part of the glaucoma. About three weeks ago, at a meeting of the St. Louis Medical Society,* Dr. H. Edward Miller reported the specific observation that the intraocular tension of glaucoma, following the injection of the sphenopalatine ganglion with carbolic alcohol, first rose 5 mm. and then dropped 17 mm. below the original tension. The effect, however, was transitory, lasting three to five days.

Dr. Otto May, in the British Medical Journal, August 21, 1912, proved that the cells of the Gasserian ganglion are very highly resistant to such a strong chemical as absolute alcohol, even though the ganglion is surgically exposed and the alcohol injected directly into its substance. At the end of two weeks such a ganglion shows almost no degenerated cells. In other words, the multipolar cells of the ganglion are not at all easily destroyed. Such a very highly resistant cell in the sphenopalatine ganglion may very readily explain such a transitory result as described by Dr. Miller.

I think that even this may not be the correct explanation of such a transitory result, because the superior cervical ganglion, as is well known, has been removed for its therapeutic effect upon the tension in glaucoma. The result of this operation is also transitory, which may possibly argue that although the sympathetic nerve supply undoubtedly has an influence upon intraocular tension, it would not seem to be the only factor.

The observation that the pain of glaucoma could be controlled from the sphenopalatine ganglion was made by Dr. A. E. Ewing in 1908, American Journal of Ophthalmology. Since that time, however, Dr. Ewing and myself have been exceedingly careful in our statements concerning the relationship of glaucoma to the sphenopalatine ganglion. Enough material has not yet been available to permit of any further statement in this connection, but the control of the pain from the ganglion seems to be definite.

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*To be published in the Annals of Laryngology, Rhinology and Otology.

NITROUS OXIDE GAS, ESSENCE OF ORANGE, ETHER AND SEQUESTRATION IN GENERAL ANESTHESIA FOR OPERATIONS IN THE UPRIGHT POSITION.

Demonstration of the Latest Model of Chair-Table.

BY THOMAS R. FRENCH, M.D.

The reception accorded the method of operating in the upright position, which was described at the last meeting of this association, must serve as an excuse, if excuse is needed, for our coming before you again to present some facts which, in part at least, have been evolved in the past year. We expressed our belief a year ago that through the upright position alone more skilful operative work could be done, that less blood was lost and that less ether was required than in other positions.

We are now able to state, as a result of the effects of the movements of the new operating table, and also as a result of the tests made in our clinic in the Long Island College Hospital of various methods of general anesthesia in the past season, that operative work in the upright position can be done with still less loss of blood, with the need of still less of the anesthetic and with less disagreeable conditions during recovery from the anesthetic.

The most pronounced effects of the smooth, gliding movements of the chair-table, even when the older methods of anesthesia were employed, have been a noticeable diminution in the loss of blood and a more satisfactory recovery from the anesthetic. This would seem to show that sudden changes in position or sudden disturbances of the body while the patient is under the influence of a general anesthetic predispose to shock, the effects of which are manifested both during the operation and during recovery from the anesthesia. After careful study of this phenomenon we became so much im-

pressed with the value of an elevation of the body from the recumbent to the upright position, for all ages of patients, without jarring, that we had the chair-table remodelled to make it possible to accommodate, from the beginning, the most diminutive of children, in order to avoid the disturbance of nervous or circulatory equilibrium which was, apparently, caused, in such cases, by lifting the body by hand to place it upon folded

Figure 1. By raising the independent seat the chair table can be adjusted for operations upon infants and small children; by raising the chair part of the mechanism to a high level the head of the patient can be brought within easy reach of the operator.

blankets so that the head could be raised to the level of the headrest. By the aid of the new attachment (Fig. 1)[1] all the movements of the body are now made with as great a freedom from jarring and disturbance of balance, with infants and very small children, as has, heretofore, been possible only with larger

[1] Figures 1, 3 and 4 show the most important improvements made in the chair-table in the past year.

children and adults; the resulting improvement in conditions leaving no doubt of the accuracy of the observation. Although, of course, we were not conscious of it during the years in which we used the ordinary chair, we believe now that the movement of lifting the patient from the table or bed to the chair, the jar and jolt of adjusting the body to the seat and back and the almost unavoidable jarring in bringing the chair

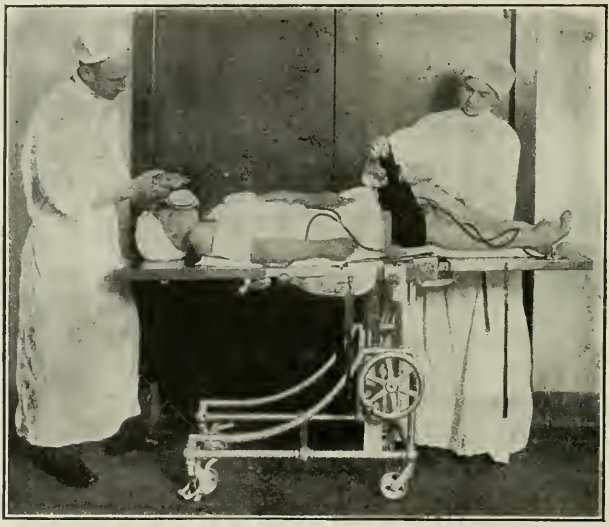

Fig. 2. The blood pressure cuffs are applied at the end of the induction stage of anesthesia. White cuffs on arms partly inflated and a moderate degree of hyperemia is apparent. Black cuffs being applied about the thighs, but not yet inflated.

to the upright position gave rise to nervous disturbances; for in no other way can we explain the improvement in the condition of the patient during and after operation since the advent of the chair-table. Our full year's experience with this instrument of precision has provided a sufficient basis for the statement that a smooth, jarless elevation of the body, without other disturbances that are absolutely necessary to bend the body from the supine to the sitting posture, must be accepted

as of fundamental importance in obtaining ideal results from operations in the upright position.

The other experiences which, in outline, we wish to describe (and this must be regarded as only a preliminary report) are especially in reference to two methods of general anesthesia.

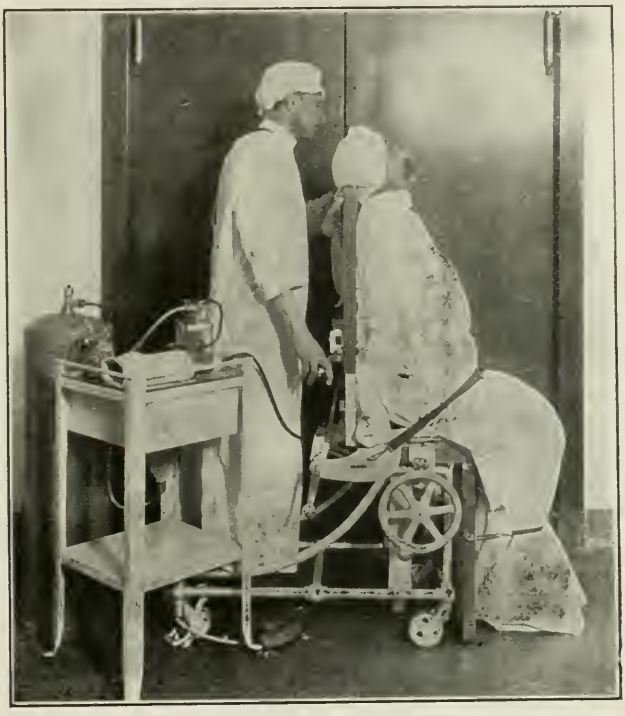

Fig. 3. The telescopic chair back can now be adjusted to any patient's back, however short or long. It can be extended somewhat further than is shown in this illustration. At full extension, as part of the table top in the horizontal position, it will support subjects of any weight.

They are: (1) Nitrous Oxide Gas or Essence of Orange Ether Sequence with, for the present, just a few words in regard to the use of Ether Vapor; and (2) Nitrous Oxide Gas or Essence of Orange Ether Sequence with Sequestration of the Limbs. At the outset it must, of necessity, be stated that the observa-

tions which follow are mainly from the standpoint of the surgeon and, therefore, not altogether that of the anesthetist.

The Nitrous Oxide Gas or Essence of Orange Ether Sequence Method.

In describing the new upright method last year we said: "The method consists in placing the patient upon the table for anesthetization in the recumbent position, and, when the stage of excitement has passed, in converting the table into a

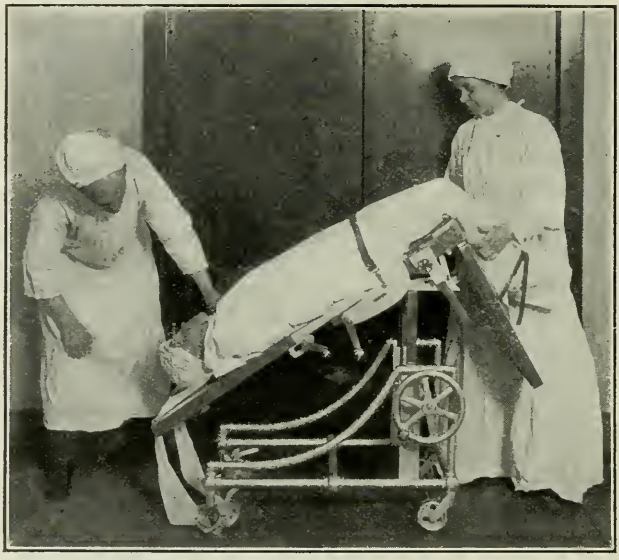

Fig. 4. Trendelenburg position; footrest of the table dropped for use with heavy subjects; pressure from the hands of an attendant or the footrest straps will keep the legs flexed and hold the body on the inclined plane without other assistance.

chair and bringing the body into a sitting position." In the light of the revelations made in the anesthetic tests of the past season, the statement of the acceptance of the stage of excitement as if it were unavoidable looked, when we saw it again recently, like ancient writing, so strongly have we been impressed, generally speaking, with its needlessness and harmfulness.

One of the most important and valuable recent contributions to anesthesia methods is the ability to omit or bridge the second stage, or stage of excitement, and, judging by the results obtained, we are deeply impressed with the desirability of attaining narcosis without struggle. This can, without doubt, be accomplished in the period of induction with nitrous oxide gas; but in our judgment it can be done with greater ease and certainty with the essence of orange. It unquestionably requires a large experience with the administration of nitrous oxide gas to enable one to dovetail it so accurately with the ether which follows that the stage of excitement will be eliminated. It can, however, be done, and when done successfully, if all other things are equal, we can safely predict the best prospect for the operation and the best condition during recovery.

The discoverer of the remarkable effects of the essence of orange as a preliminary to ether, Dr. James T. Gwathmey, of New York, has demonstrated its possibilities in our clinic and has proved to our satisfaction that with it the administration of ether is made far less disagreeable and that it greatly assists in the reduction of shock by bridging the stage of excitement. We have seen the inventor of this method use it repeatedly with perfect success, the patients sinking into complete anesthesia as a child falls to sleep. Our observations thus far have brought the belief that an anesthesia conducted in that way up to the time of raising the body to the upright position is a contributive factor in reducing hemorrhage, also in reducing the quantity of the anesthetic and in shortening and modifying the anesthetic after-effects.

In the testing work in anesthesia, more particularly that carried out during the past season, the writer has had foremost in mind the great need of reducing shock. It is generally conceded that the child is more susceptible to shock than the adult, due in some way, no doubt, to the fact that the child is anatomically and physiologically different from the adult. The tests have, therefore, been applied especially to children in the controlling of hemorrahge and in the administration of anesthetics. We find no variance from the belief among surgeons who have given careful thought to their work upon

infants and children that the losing of blood is a matter of the greatest import to them and that all means should be used to prevent hemorrhage in operating upon them. And we are now convinced that shock from the loss of blood and from the anesthetic can be materially reduced by the *manner* of administering the anesthetic.

According to the observations of the writer of this paper, there is, irrespective of all other conditions, a well defined and never failing relationship between the degree of skill in which a patient is anesthetized in the upright position and the amount of hemorrhage which occurs during the anesthesia, for there seems to be no question but that hemorrhage is reduced if the anesthetic, from the beginning, is smoothly administered, the second stage omitted and the patient brought into surgical anesthesia without jarring or body disturbance of any kind. The uniform employment of helpful mental suggestion by every individual in contact with the patient up to the time of the induction of anesthesia, to assist in preventing an excessive discharge of nervous energy through fear—which is one of the elements in the "anoci-association" of Crile—the administration of morphia to patients who display a marked degree of apprehension; an anesthetizing room free from an atmosphere of excitement and from unnecessary noise; the preliminary use of nitrous oxide gas or the essence of orange and the drop method of ether with an open inhaler and later with an Allis inhaler, will insure, at the hands of a competent anesthetist, the prolongation of the anesthesia after the body reaches the upright position, a reduction in hemorrhage and also a reduction in the disagreeable sequelae.

We have found the closed method of administering ether vapor, or, more properly speaking, ether insufflation, devised by Dr. William C. Woolsey, of Brooklyn, of great value in maintaining anesthesia in long operations. It must be remembered that anesthesia is maintained, for a time at least, after the body has been raised to the upright position, by the position alone. If the induction has been satisfactory the anesthetic not only should be, but, in most cases, must be diminished in quantity, or withdrawn, as soon as the upright position has

been attained, to prevent the narcosis from becoming too deep for safety. When the reflexes begin to reappear the anesthesia can be continued by the occasional administration of the vapor through the mouth. (We exclude the nasal method, at least for the present, because it is still a question in dispute as to whether the passage of a stream of ether vapor under pressure through the nose is not capable of producing irritation of the various accessory sinuses.) The fact that only a half, or less than a half, of the usual quantity of ether is required to maintain anesthesia with this method should not deceive us into believing that we get only a half of an anesthesia with it, for it is, in reality, a full one, and while the patient is in the upright position a continuous flow of vapor under pressure is, as a rule, not permissible. The only way in which ether vapor can be administered continuously while the patient is in the upright position is with the admixture of oxygen, but this combination, while producing a pleasing color of the face, increases hemorrhage. It is, however, always advisable to have the oxygen tank attached to the vapor apparatus to quickly relieve a narcosis if it should become too deep.

One of the results to be contended for is the avoidance of vomiting after operation to reduce the chances of infection of the wounds in the nose and throat. Of course, if a considerable quantity of blood has been swallowed, vomiting is desirable to prevent irritation of the gastro-intestinal tract, and, fortunately, most of it is usually ejected without therapeutic assistance. Therefore, if the diet table before operation is properly carried out, the less the loss of blood, the less the need of vomiting, and, the less the amount of the anesthetic required for anesthesia the less the chances of vomiting both during operation and afterward.

There is, we believe, no more contributive factor in securing a rapid and an uneventful recovery than a well conducted anesthesia. That we are now in a position to be assured that an anesthesia can be well conducted, in the large majority of cases, through the co-operation of an accomplished expert anesthetist, is one of the delightful and satisfying outcomes of specialization in medicine. The day is probably near at hand

when, except in emergencies, every operation of importance will be performed with the assistance of an anesthetist who is an acknowledged expert in his field.

Nitrous Oxide Gas or Essence of Orange Ether Sequence with Sequestration of the Limbs.

This method consists in anesthetizing the patient in the manner just described and at the same time producing congestive hyperemia of the limbs by means of inflated blood pressure cuffs about the thighs and arms (Fig. 2), or thighs alone, to still further reduce the amount of blood in the head. Sequestration of the limbs was, we believe, first suggested by Dr. R. H. M. Dawbarn, of New York, in 1907, to reduce hemorrhage in operations upon the head. In our recent trials of various methods of anesthesia, Dr. Gwathmey advised a test of the Dawbarn method to determine its value in reducing hemorrhage in operative work in the nose and throat. He thought it would be a safe procedure if used with the new table. While contemplating a trial of sequestration we confess to having had a feeling of trepidation lest something should go awry and especially because of the warning given us by one of our ablest clinicians who thought we would be likely to produce artificial syncope which might be difficult to control. We, therefore, gave the suggestion of such a test long consideration, during which we recalled, among other things, the not inconsiderable number of times in which, on freeing the patient from the chair used in the older method, we had found both arms deeply congested, indeed quite black, from the careless adjustment of the shoulder bandage. Those incidents, however, seemed to have caused neither disturbance during the operation nor harm in any form afterwards. This gave us a considerable degree of assurance that the reduction in the amount of body tissue to be anesthetized would be free from danger. We relied, however, largely upon the ease and rapidity with which the new table could be tilted in the opposite direction for resuscitation, if conditions should require it. After beginning with a light pressure of the cuffs upon

the thighs alone, which was afterwards in other cases gradually increased, we attained at the end of the first operating clinic in which these tests were made as high a degree of pressure as we are using now; but it was not until a later occasion that we felt justified in employing the inflated rings upon all four of the limbs at the same time. In the first case in which the trial was made it was found that there was a marked diminution in the amount of blood lost and also that the anesthesia was prolonged after removing the inhaler at the beginning of the operation to a much greater extent than we had ever known it to be by any other method. And with very few exceptions—in which the conditions were unfavorable—the same experience has been repeated in every case in which the method has been used. Dr. Gwathmey guided the anesthesia and controlled the blood pressure cuffs in all the early cases in this test, the subsequent cases having been supervised by the hospital expert anesthetist, Dr. A. F. Erdmann.

We said in our paper of a year ago that two of the most important reasons for preferring the upright position were a reduction in the amount of the anesthetic required and a reduction in the amount of blood lost during operation. The sequestration method, in association with the upright position, which has been carried out in 72 cases during the past season, reduces still further the amount of the anesthetic required and still further the loss of blood. That the amount of the anesthetic is reduced is proved by the fact that full anesthesia has often been maintained for fifteen or twenty minutes after the body was brought to the upright position and the inhaler removed. Indeed, after beginning the operation, in most cases in which this method of anesthesia has been used, except for his assistance in other ways, we have been able to dispense with the services of the anesthetist. Which is only another way of saying that during relatively short operations the further administration of the anesthetic has not been needed.

In quite a number of cases the quantity of blood lost during the removal of adenoid growths and faucial tonsils (and we use those operations in illustration because the amount of blood usually lost in them is so well known) while sequestration was

being applied, was believed to be less than a drachm. In many of the cases the blood loss, in operations in either the nose or throat, could only be measured by the appearance of the sponges; in most cases it was but slightly in excess of that and in none was the bleeding as free as in the average case of similar operative character under the older methods of anesthesia. It may, therefore, be said that the average blood loss with the sequestration method, whether applied to arms and legs or legs alone, is far below that which occurs without sequestration, and that certain operations which with the ordinary methods are usually attended with a large loss of blood may be rendered practically bloodless by the use of sequestration in the upright position.

We had thought of the possibility of secondary hemorrhage being occasioned by the sudden release of the locked up quantity of blood at practically the same moment that the body was returned to the horizontal position; but, happily, there does not seem to be reason for apprehension on that score, for after the operations were finished and the bleeding arrested, there was no further hemorrhage in this series of cases.

We have already spoken of the careless adjustment of the shoulder bandage and desire to call attention to it again in connection with sequestration. If the bandage is adjusted before the body settles into the sitting posture and is drawn too tightly the patient may be literally hung up by the axillae, when of necessity great pressure must be made upon the axillary arteries. This has not occurred during our tests because the drill of the assisting staff made it quite impossible; but should it occur while sequestration was being applied to both arms and legs, we can imagine the face assuming a hue which would suggest the propriety of tilting the body into the Trendelenburg position. As the "upset" movement can be quickly made with the chair-table, the conditions, in even such an extreme case, could be easily remedied.

Sequestration would have been used in a much larger number of cases had it not been necessary to make each test with deliberation in order that all conditions during operation could be noted, and also to permit the following up of the

cases to determine whether injury to the nervous tissues or the musculature of the limbs could have been produced. The amount of pressure made with the cuffs varied from that needed to effect a slight diminution in, to that needed to produce a complete obliteration of, the pulse in the anterior tibial and radial arteries, and we feared that the firm pressure required to cause an obliteration of the pulse might produce injury to nervous structures; but in none of the cases could any history of untoward after effects be elicited. It is quite possible, perhaps even probable, that this can be explained by the comparatively wide distribution of the pressure and the fact that it was made with a flexible air bag. The pressure was maintained, in all but the earliest cases, from the end of the induction stage of anesthesia to the time of completing the operation.

The only patient operated upon since the series of tests of sequestration was begun who presented symptoms after operation which suggested a previous injury to the legs was a very delicate child of five years, about whose thighs the cuffs were applied, but which were not inflated. As the patient was an extremely delicate piece of human mechanism and the test was in its erliest stage we regarded it as unwise to expose it to what might seem to be something of a risk. It would, therefore, appear that we just escaped the reputation of having had one harmful effect from this method of anesthesia.

To be able to administer a smaller quantity of an anesthetic and obtain a full anesthesia; to see the patient enter the anesthetized state without the stage of excitement; to be able to stop the administrtion of the anesthetic when the body is brought to the upright position and yet have the anesthesia prolonged enough to permit relatively long operations to be performed; to seeure a greatly lessened loss of blood; and to insure a reduction in, and in many cases an almost complete abolition of, the disagreeable after effects, is, to say the least, something of a gain, and in a large proportion of cases, with the co-operation of a skilful anesthetist, all the above results are obtainable.

Our controlling thought in this work has been, and is, to

reduce the necessity for "rough" surgery and also to assist in the establishment of improved conditions from the standpoint of the patient. Crile—than whom none have done more scientific and humanitarian work in regard to shock—says in speaking of "anoci-association": "Now, I am ready to admit that to accomplish this it is necessary to especially train the nursing department, the hospital staff, the anesthetist, and, above all, to train one's self to act with the greatest precision and only in the interest of the patient, and also I must admit that it immensely increases every one's labors." Such work does require time and much careful attention, but is it not time well spent and is it not in accord with the high ideals of our craft? A number of general surgeons, after viewing this kind of work, have expressed their doubts of its necessity, "for," they have said, "we rarely see surgical shock." They evidently think that nothing much counts short of profound shock. Well, there can be no doubt that many things occurring before and during operation contribute to shock, and the cumulative effect of many small shocks may be, and we believe often is, an exhausting discharge of nervous energy. If these premises are correct, then, manifestly, it is our plain duty to make note of and carry out the details and so contribute to the amelioration of the patient's distress. *Surgery has recently made a distinct advance in that, largely through "anoci-association," operations have been still further robbed of their terrors.*

Discussion.

Dr. John O. Roe, of Rochester: The question of shock, emphasized by Dr. French in describing his admirable methods of handling patients during operations, with his very ingeniously devised chair, is a very important matter, since shock should be eliminated as far as possible in all cases. This is particularly the case with children, for any terrors they may experience form a lasting impression not easily gotten rid of. This fact can be more fully appreciated by reading Dr. Crile's paper on Anocia. In children and highly nervous adults the dread of an operation and the shock attending it can be very largely obviated by a good sized hypodermic dose of morphia a short time before the operation. The morphia also has a marked effect in controlling hemorrhage.

Dr. Arthur W. Watson, of Philadelphia: I wish to re-

call the fact that the late Dr. Harrison Allen was an advocate of the upright position in operating on the throat and nose, and during the latter part of his life used that position in all of his operations. Our recently deceased fellow, Dr. Bliss, as an assistant to Dr. Allen became familiar with the method and always used the upright position in his tonsillectomies.

Dr. George L. Richards, of Fall River: I have operated on patients in the upright position for a number of years with a minimum amount of hemorrhage and had no unfavorable results following the operation which could in any way be charged to the upright position.

Dr. B. R. Shurley, of Detroit: The modern views at the present time regarding operations upon cases with well marked cardiac lesions are at variance. I would ask what experience any of you have had with cases of this kind when operated on in the upright position when they had old cardiac lesions or advanced cardiac disease.

Dr Thomas H. Halstead, of Syracuse: I have used the table presented by Dr. French and with much satisfaction. In the upright position there is less hemorrhage, but I have not used the sequestration method. I am very much pleased with the use of the table.

Dr. Christian R. Holmes, of Cincinnati: I would like to ask Dr. French what his opinion is as regards the upright position compared with the horizontal position.

Dr. James E. Logan, of Kansas City: Do secondary hemorrhages occur, that is, hemorrhages three, four or five hours after the operation?

Dr. Joseph H. Bryan, of Washington: I believe in these adjuncts to anaesthesia, especially where we are looking for serious hemorrhages in the upper air passages and particularly from the upper and posterior sinuses. I believe that many cases passing off on the table could be carried along, saved from death and from alarming hemorrhages by the use of these adjuncts.

Dr. Harmon Smith, of New York: I have seen Dr. Dawbarn operate upon a case in which considerable bleeding was to be expected, but by the employment of sequestration of the lower extremities little or no hemorrhage resulted. I believe that in the suction employed for the removal of blood in the naso-pharynx during the time of removal of tonsils and adenoids, considerably more is drawn away than would otherwise happen, and at times in anaemic children this loss of blood is of serious consequence. If one could reduce the amount of hemorrhage by sequestration it would lessen the obstruction to the operative field and

also be of material benefit toward the rapid recovery of the patient subsequent to operation.

Dr. French (in closing): I will say that, as the programme is behind time, I will be obliged to answer many of the questions in a general way and omit much that the discussion tempts me to say. My experience in a very large number of cases has been that the patients do quite as well in all ways in the upright as in the horizontal position, and while I can not recall having used the upright position for patients with dilated hearts, I have frequently used it for patients with valvular defects. I have, in a few instances, found it desirable to "upset" the body in such cases, but have never been seriously embarrassed. Now that the body can be so easily placed in the Trendelenburg position, I am not sure that I have not occasionally tilted the patient head downward to relieve a venous congestion which before the table came into use was not regarded.

There is no more likelihood of secondary hemorrhage occurring after operations in the upright position than in any other position.

Sequestration reduces shock by reducing the quantity of the anesthetic required and also by reducing hemorrhage, but does not increase the chances of secondary hemorrhage.

Parts of the subject brought forward by a number of the speakers were discussed pretty thoroughly at the last meeting, and because of the limited time at our disposal should, perhaps, not be elucidated now.

The object in making the table top as narrow as it is was to make it easy for the anesthetist and assistants to readily reach all parts of the body when the subject was a small child. I have, however, about decided that Dr. Halstead's criticism is valid and the top should be made a trifle wider.

The revelations made by the chair-table have been a great surprise to me. I had no idea when a means was devised to raise the patient from the horizontal to the upright position in a jarless way that it would result in such a distinct improvement in the patient's condition both during and after operation. It was that, more than anything else, which has led me to a further appreciation of the value of Crile's brilliant experimental work in regard to shock; a value which he could not but believe surgeons in general have not yet fully awakened to. When we become impressed with the truth of Crile's definition of shock that it "represents the sum total of all impulses that reach the brain by any path," we will, I believe, contribute in a much more active way to the development of a new era in surgery.

THYROTOMY FOR CANCER OF THE LARYNX, WITH REPORT OF ELEVEN CASES.

BY D. CROSBY GREENE, JR., M.D.

In his recent paper on Laryngectomy for Cancer, Crile,[1] in discussing the surgery of the larynx, confines himself to the operation of total extirpation of the larynx. While this operation is appropriate for cases of extrinsic cancer and advanced intrinsic cancer, there is a large proportion of early intrinsic cases in which there is a reasonable hope of cure by the comparatively slight operation of thyrotomy, originated and advocated by our distinguished fellow member Dr. Solis-Cohen[2] in the year 1867.

The basis for this claim rests, in the first place, on anatomic grounds. The favorite site for epithelioma of the larynx as appears from statistics of others and from my own series is on the vocal cord. In this region the conditions are peculiarly favorable to slow growth and extension, owing to the dense fibrous structure of the cord and its meager lymphatic supply, both as regards the number and size of the lymphatic vessels.

In a paper presented as candidate's thesis to his association in 1906[3], I reported the results of an investigation of the lymphatic drainage of the larynx by means of submucous injections of methylene blue and mercury. These results were confirmatory of those reported by Cuneo[4], Most[5], and others, showing that the network of lymphatic vessels which extends beneath the mucous membrane throughout the interior of the larynx is rich in the number and size of the vessels in the supra-glottic region, relatively poorer in the subglottic portion, while on the vocal cords themselves the vessels were very small and widely separated. These anatomical facts account

[1]Crile. Laryngoscope, Dec., 1912.
[2]Solis-Cohen. N. Y. Med. Record, 1867, p. 218.
[3]Greene. Transact. Am. Lar. Assn., 1906, p. 63.
[4]Cuneo. Gazette des Hopiteaux, 1902, No. 141.
[5]Most. Anat. Anzeiger, 1899, p. 387.

for the slow growth and late involvement of the cervical lymph nodes in cases of epithelioma of the cords, and furnish an argument for the possibility of cure in early cases by the operation of thyrotomy and excision of the growth with a wide

TABLE OF CASES.

	AGE	SEX	SITE OF GROWTH	HISTOLOGICAL DIAGNOSIS	DATE OF OPERATION	RESULT
Case I.	62	Male	Left Vocal Cord	Squamous Cell Cancer W. F. Whitney	August 27, 1905	Death from operation
Case II.	61	Male	Right Vocal Cord	Squamous Cell Cancer C. C. Simmons	January 1, 1906	Recurrence in 2½ years Death in 3½ years
Case III.	58	Male	Left Ventricular Band and Left Vocal Cord	Squamous Cell Cancer W. F. Whitney	August 5, 1907	No recurrence May 1, 1913
Case IV.	28	Female	Left Vocal Cord	Squamous Cell Cancer W. F. Whitney	May 11, 1909	No recurrence May 1, 1913
Case V.	54	Male	Left Vocal Cord	Squamous Cell Cancer W. F. Whitney	October 31, 1910	No recurrence May 1, 1913,
Case VI.	60	Male	Extensive growth, involving both sides of Larynx	Squamous Cell Cancer	November 10, 1910	Recurrence in 1 month Death in about 6 months
Case VII.	52	Male	Arytenoid	Squamous Cell Cancer H. A. Barnes	November 1, 1911	No recurrence May 1, 1913
Case VIII.	60	Male	Right Vocal Cord	Squamous Cell Cancer W. F. Whitney	November 2, 1911	No recurrence May 1, 1913
Case IX.	60	Male	Left Vocal Cord	Squamous Cell Cancer H. F. Hartwell	April, 1912	No recurrence one year after
Case X.	53	Male	Extensive growth occupying left side of Larynx and upper Trachea	Squamous Cell Cancer H. F. Hartwell	June 26, 1912	Recurrence in 1 month Death in 6 months
Case XI.	65	Male	Left Vocal Cord	Squamous Cell Cancer W. F. Whitney	July 15, 1912	No recurrence May 1, 1913

margin of healthy tissue. This theoretical contention is supported by the results of the operation by numerous operators both in this country and abroad, so that at the present time it is almost universally recognized as the proper procedure for the treatment of early intrinsic cancer of the larynx. Even Gluck[6], whose unrivalled experience with total laryngectomy and recently published remarkable record of sixty consecutive cases without operative mortality naturally inclines him to the side of radicalism, advises thyrotomy in early intrinsic cases.

In presenting this report of my own cases, which is so limited, as to number as well as to the time elapsed after operation in many of the cases, that of itself it deserves little weight in determining the value of the method, my object is to call attention to certain details of the technic which to my mind have an important bearing on the immediate and later results of the operation. The steps of the operation as I have done it in the last five cases are as follows:

1. *Narcosis.*—Ether by inhalation preceded one hour before operation by morphine gr. ¼ and atropine gr. 1-150.

2. With the head slightly extended a median incision is made, extending from the lower border of the hyoid bone to the lower border of the cricoid cartilage. This incision is carried down through the pre-thyroid muscles until the thyroid and cricoid cartilage and crico-thyroid membrane have been definitely exposed.

3. A 1 per cent solution of cocaine is injected with a hypodermic syringe through the crico-thyroid membrane into the cavity of the larynx.

4. The patient is now put in the Trendelenburg position and a thick pad placed under the shoulders to bring the larynx into greater prominence.

5. The crico-thyroid membrane is next incised in the median line, and through this incision a swab of 10 per cent cocaine is introduced and applied to the laryngeal mucous membrane.

6. The thyroid cartilage is, after a pause of 5 minutes, divided from below upwards. In young subjects this may be

[6]Gluck. Jahreskurse f. Artz. Fortbildung, No. II., Vol. III., p. 20.

done with a knife, but since in the majority of these cases the cartilage has become ossified with age, it is usually best to use for this purpose strong curved scissors with dull points.

7. The thyroid wings are now widely retracted and a careful examination of the growth made under good illumination with head mirror or headlight.

8. Beginning at the free margin of the thyroid cartilage on the affected side in front of the growth the internal perichondrium is elevated from off the cartilage with a sharp elevator from before backwards to a line well behind the limits of the growth as well as above and below it.

All the soft structures in the vicinity of the growth are thus freed from the underlying cartilage.

9. Two parallel horizontal incisions are now made with scissors above and below the growth, leaving a ½ inch margin of safety. These incisions are carried about ½ inch back of the posterior limit of the growth.

10. The growth, with its surrounding tissue, is now entirely free, excepting for its posterior attachment. A wire snare passed over this mass of tissue is used to sever the posterior attachment. The edges of the thyroid cartilage are next approximated and held together by S.W.G. sutures passed through the overlying soft tissues. A rubber drain is left in the lower end of the wound for twenty-four hours.

I have found the narcosis by ether preceded by morphine and atrophine to be very satisfactory. The ether is administered by inhalation through the natural passages in the usual manner and the patient carried to a profound anesthesia until the larynx is opened. The remainder of the operation is completed before the patient comes out of his anesthesia, which is prolonged by the sedative effect of the morphine and the local anesthesia of the cocaine. In only one case was it necessary to administer more ether after the larynx was opened. In this case a sponge, saturated with ether held over the open larynx for a few minutes, was sufficient to prolong the anesthesia. Etherization by rectum was tried in my first case. Its obvious advantages during operation were unfortunately counter-balanced in this case by uncontrollable bloody

diarrhoea, collapse and death within twelve hours after operation. I have, therefore, not given this method another trial.

The value of atrophine and cocaine in the prevention of shock in operations on the larynx has been fully demonstrated by Crile.

In spite of Jackson's warning against the use of morphine in these cases, I have never seen any ill effect from its use. His point is that it deadens the cough reflex, which is "the watch-dog of the lungs." In practise I have found that the maintenance of the extreme Trendelenburg position during operation and keeping the foot of the bed moderately elevated for three to six hours after operation, was sufficient to prevent drainage downward. After that time patients tend to cough too much rather than too little.

There was no inhalation pneumonia in any of my cases. In only one was there an acute bronchitis following the operation. This case had a chronic bronchitis of several years standing with considerable expectoration. When his larynx was opened a stream of grayish bronchial secretion was seen flowing down from the trachea over the field of operation.

In my experience tracheotomy, excepting in cases of dyspnea, has proved to be unnecessary. It prolongs the operation without serving any useful purpose. In the last seven cases I have practised it only once.

The most important part of the operation is the actual removal of the growth. This should be done without at any point cutting into the growth, a proceeding which, as Crile insists, is fraught with danger of spreading the disease. In the three cases of recurrence in my series I know that this fault was committed. Now the special value of the method which I have described lies in the fact that the sub-perichondrial separation of the growth from the cartilage as advocated by Solis-Cohen,[7] being accomplished without any bleeding to obscure the surface of the tumor, the operator is enabled to make the two horizontal incisions above and below the growth accurately and with speed. The completion of the operation by the use of the snare facilitates the last step and diminishes

[7]Solis-Cohen. Transact. Am. Lar. Assn., 1907, p. 150.

the amount of oozing from the posterior part of the wound, where it is apt to be most profuse and obstinate. If the growth is seen in the preliminary examination to extend forward to the anterior commissure or near it, there is danger of cutting into it if the thyroid cartilage is divided in the median line. In such a case it is better to make the division parallel to the median line, but a little to one side, even though this involves injury to the sound cord.

After Care. Immediately after operation the patient is put to bed with a flat pillow, and the foot of the bed raised to favor the expulsion of blood and secretions from the larynx and trachea. A pint of normal salt solution is given by rectum every six hours for the first twenty-four. At the end of this time the patient can usually swallow liquids. He is first tested with a little sterile water, and if this goes down without making him choke, feeding by mouth with sterile liquids is begun. In most cases the patient can sit up on the third day and walk about at the end of a week. This was true of all but two of my last cases who lived.

The only death from operation was in my first case, which I have attributed to rectal etherization.

The healing of the external wound occurred, in six of the cases, by first intention. In the remaining four there was a varying amount of local sepsis, with delayed healing. In three there was necrosis of a bit of the thyroid cartilage. In one case this was coughed up by the patient one month after operation.

In all the cases in which the laryngeal wound was closed without leaving a tube in the trachea, there was more or less emphysema. This was marked in only one case, in which the air extended up over the chin and up to the eye on one side. This condition was relieved by the removal of a stitch from the lower end of the wound and allowing an external vent to the air in the larynx.

In all cases there was eventual healing with an insignificant external scar.

In one case there was an extraordinary acceleration of the pulse, without accompanying rise in temperature, which came

on the second day after operation and continued to increase for twenty-four hours after reaching 140 to the minute. Then it began gradually to subside, but it did not become normal for ten days. Crile mentions the occurrence of this phenomenon after laryngectomy, and attributes it to vagitis from injury and infection of the pneumo-gastric nerve. In thyrotomy direct injury to the pneumo-gastric is hardly possible. In the case to which I referred there was present an unusually large and highly placed thyroid isthmus, which was cut into and retracted downwards with some force. It occurred to me that in this case the acceleration of pulse might have been a manifestation of hyper-thyroidism due to injury of the gland.

In regard to the question of recurrence, my statistics are too meager in number and time elapsed after operation for drawing positive conclusions. Of the ten who survived operation recurrence occurred in three cases. In one of these, Case II, recurrence came two and one-half years after operation. In the other two, Cases VI and X, recurrence was evident one month after operation. In all of these cases the growth was cut into in the course of the operation. In Case II, a favorable case, this was due to imperfect technic, while Cases VI and X were advanced cases suitable for laryngectomy.

In the remaining seven cases there has been no recurrence to date. Case IV, the only woman in my series, was operated upon by Dr. Coolidge and myself at the Massachusetts General Hospital in May, 1909. This patient was a domestic, who, at her last visit to the hospital one and a half years ago, was on the point of going home to Ireland. She was then in excellent condition, without evidence of recurrence, and had a fair voice. A note just received from her states that she is well and having no trouble from her throat. Case III has gone without recurrence for nearly six years. Case V two and a half years; Cases VII and VIII, one and a half years; Case IX, one year, and Case XI, nine months.

In conclusion, I would emphasize the importance of a proper selection of cases. Not all intrinsic cases are suitable for this operation. When the growth is so extensive, even when confined within the cavity of the larynx, that the larynx cannot be

opened without cutting into the growth recurrence is not only possible but probable.

As far as they go, the figures from my series confirm the brilliant results of Cohen, Semon, Butlin, Jackson, Schmiegelow, St. Clair Thomson and others in obtaining lasting cures in a large percentage of their cases. Their results may be repeated by attention to the essential points, namely, a proper selection of cases and careful technique.

Discussion.

Dr. Chevalier Jackson, of Pittsburgh: I disagree with Dr. Greene in the statement that the value of thyrotomy for cancer in the larynx is generally recognized. Unfortunately, it is not very generally recognized, because in some quarters thyrotomy for cancer in the larynx has been disappointing. The reason for the disappointment is simply that unsuitable cases have been operated upon. My own cases of thyrotomy have convinced me that nowhere in the realm of malignant disease are such satisfactory results obtained from operation as in thyrotomy for cancer of the larynx. There is practically no operative mortality. In twenty-five of my cases twenty-three were free from recurrence for periods varying from one to fifteen years. Vocal results are excellent in all cases where no injury has been done to the cricoarytenoid joints. This is an important point which I was the first to demonstrate; namely, that the formation of adventitious bands depends upon activity of the cricoarytenoid joint in pulling out a new band from the cicatricial tissue. Growths involving the arytenoid joint are not, anyway, suitable cases for thyrotomy because it is only in the anterior growths of very small extent situated intrinsically that yield permanent cure. Laryngectomy is justifiable in a very limited number of cases, and the results in no way compare in efficiency with the results of thyrotomy. In regard to anesthesia, I have been delighted with the intratracheal insufflation method of Elsberg with ether. I prefer not to sew up the wound, but allow the cartilages to be united with fibrous tissue while the wound gets packed open. After fibrous union of the cartilage is complete the superficial wound is then allowed to fill up, but no stitches are used.

Dr. Cornelius G. Coakley, of New York: I am very much interested in the paper just read and I agree with the last speaker that a great many cases of malignant disease of the

larynx come to us too late for any operative work. We have some total laryngectomies but cannot give end results. However, in selected cases thyrotomy offers the best hope.

Morphine is a very proper agent to give, and makes the anaesthesia much easier for the anaesthetist to give; anaesthesia as administered by an expert, as Dr. Bennett, of New York, gives but little trouble and the patients as a rule are out of the anaesthesia as soon as they are through with the operation.

Another point I think will work out well in practise, and a method employed by Dr. Erdmann, of New York, is to place no sutures at all in the thyroid cartilage after opening up and separating the larynx widely. The healing has been good in every case. I think that most of you will find it is not necessary to place sutures in the thyroid; the sutured fascia holds the parts together sufficiently well.

Dr. Emil Mayer, of New York: Too much stress cannot be laid upon the inadvisability of wounding the cancerous growth of the larynx unless we are prepared to go ahead and do some radical operation. I will cite a case in point. A man came to me one year ago with the diagnosis of cancer of the larynx. I told him that I thought the condition was cancerous, but I would not take a piece from the growth for examination by the pathologist unless an immediate operation was consented to if the diagnosis was confirmed. The patient's business was such that he could not give this consent and, therefore, I declined to remove a piece of the growth. This man's business took him to Europe and he was away two months. During the time away he had consulted three colleagues and he returned with the following diagnosis: Tuberculosis of the larynx; tuberculosis of the larynx; cancer of the larynx. I still insisted upon the diagnosis being cancer of the larynx and an operation was done. Epithelioma was found; the man died subsequently. I contend that harm will result from any incision into the suspected growth. There is no question in my mind as to the correctness of the diagnosis I had made of cancer of the larynx.

Dr. William L. Ballenger, of Chicago: I have operated on only a few cases of cancer of the larynx, probably fifteen (15) in all, and most of them came to me well advanced. I only recall three (3) such cases in which I did thyrotomy. On one patient the operation was done three (3) years ago and he is still alive and without any signs of recurrence. On the second case hemi-laryngectomy was done; after three months' observation, I did a total laryngectomy. This patient was a man thirty-three (33) years of age. The diagnosis of specific dis-

ease was made and iodide of potassium administered. The microscopical report came back, carcinoma of the larynx. Operation was done, and the man is still living and in apparently perfect health. A peculiar feature of this case was that he had enough voice left to be understood and had been advised to go and have his throat treated. He replied that he was supposed to have no voice "because his throat had been taken out." Thyrotomy was performed on the third case two years ago. I saw the patient two months ago and there were no evidences of any recurrence of the growth and the man was apparently in good health. All the other cases were advanced cases and all but two are dead. Two died within one week after operation. Some lived a year or more and died within one week after operation. Some lived a year or more and died from recurrences. I wish to emphasize the importance of early diagnosis in these cases of cancer of the larynx.

In regard to the location of the growth in the cases operated upon, in three (3) it was located upon the false cord. I do not believe that we are yet prepared to say that laryngectomy is not a suitable operation in these cases; this operation, I think, should be tried. Dr. Crile operated upon a man with advanced extrinsic growth who had remained well afterward for several years. There are some cases on whom a total laryngectomy is performed which do get well. The percentage is small, but large enough to warrant its careful consideration.

With regard to the use of anaesthesia, I am satisfied with the use of the local anaesthesia, and I make this decision after trying both. This can be used without pain or discomfort.

With regard to the incision into the cartilage, this I do with Jackson's scissors.

Dr. Ballenger: Of my fifteen (15) cases there were twelve (12) laryngectomies. I never did a preliminary tracheotomy in these cases. Dr. Keene has advised thyrotomy without a preliminary tracheotomy.

Dr. George L. Richards, of Fall River: I will refer to a patient I have seen one and a half years ago. An examination of the larynx showed a warty growth about the junction of the middle and anterior folds, and the pathological examination of the piece removed brought back the report that it was non-malignant. The examination of the next piece removed brought back the report that it was malignant. Everything was gotten clean for operation. On April 9, 1912, operation was performed and only one week ago the patient came to see me and there was absolutely no recurrence of the growth. He

had a very small false cord and no true cord. In this case I took off one-half the arytenoid and the results have been fairly good, but he has a hoarse voice and rather an anemic look. For weeks after the pulse was very rapid, and whether this was due to the injury to the thyroid or not I do not know. This apparently was due, however, to his cancerous cachexia. There has been no return of the growth. The fistula resulting has totally closed up. He lost a considerable amount of flesh.

I have one other case still progressing. I did a tracheotomy on an apparently hopeless case in which both cords were involved in the process. The tracheotomy was called for because of the difficult breathing. It is now three years since the operation. The man is a bartender to-day and is doing his work right along. The other day the tracheotomy tube came out and we thought he would die before getting it replaced. He lost flesh for two or three months. Three years have now elapsed since the operation.

Dr. Smith, of New York: I have recently had under observation an old man with cancer of the tongue, whose condition was absolutely beyond help from operative measures. In consultation with Dr. Beebe, who is an authority on autogenous vaccine injections, it was determined that colloidal copper should be employed by intravenous injection. This method was performed until all available veins had been used up, the last injection being made into the internal saphenous vein. At this time the patient rebelled against further injections, but the progress of the growth unquestionably was retarded. Colloidal mercury was now employed in very much smaller quantities and several deep sloughs in the tumor resulted, showing that some material changes had taken place in the cancer itself. At this time, several months after the admission of the patient into the hospital, he became rebellious and refused further treatment and left the hospital. He is now, however, able to return to the clinic three times a week for observation and has gained in weight and strength, there having been apparently no increase in the progress of the tumor. As nothing definite can be determined by the progress of one case, the relation of these facts may be taken only as an index to what may be possibly accomplished if employed in the early stages of cancer.

With regard to the thyrotomy cases there is often bruising and tearing of the perichondrium, which later results in the formation of granulations which prolong the recovery of the case. General surgeons are prone to disregard the minute detail so essential for the proper care of the larynx and pay too little attention to the preservation of the arytenoids or

small parts of the true cords, which if left insures to the patient the probability of greater and better voice production. I would advise that only sharp instruments should be employed in the removal of the growth from the larynx, and that a clean wound would be left, from which there is little or no danger of oedema. Preliminary tracheotomy may be obviated in the majority of cases, especially if the Trendelenburg position is employed.

Dr. Christen Quevli, of Tacoma; by invitation: The reason why so many cases come to the specialist too late for successful operation is the failure of the general practitioner to diagnose the cases earlier. It seems to be the prevailing idea among the general practitioners that besides continued hoarseness you should also have pain. The reason for this is that many of the prominent authors give pain as a symptom in intrinsic cancer of the larynx. I have worked for eight months in Chiari's clinic in Vienna, and neither there nor in my own private practice have I ever seen a case of intrinsic cancer in which the patient complained of pain.

Dr. William E. Casselberry, of Chicago: I was glad to hear the essayist state plainly that preliminary tracheotomy is unnecessary in thyrotomy for cancer of the larynx. Regarding adrenalin, I think it aids vision within the divided larynx by helping to control whatever diffusion of blood, even though slight, may accompany the mucoperichondrial incision. I would like to ask Dr. Greene why he withdraws its use. In one of my cases of thyrotomy in a child for papilloma, serious cicatricial stenosis followed and resulted fatally by reason of an accidental displacement at night of the tracheotomy tube. I favor the operation of thyrotomy for cancer whenever seemingly adequate, rather than laryngectomy and will compare two cases that occurred in my practise. In one after thyrotomy the patient lived ten (10) years in full enjoyment of his faculties and without recurrence of the cancer in the larynx, death took place finally from cancer of the liver; in the other, after total laryngectomy, the patient, though still without recurrence of the cancer, is melancholic because the mutilation and vocal disability render him dependent for support, which is apt to be the case after a total laryngectomy. I speak of this because I have noticed in general surgery a pronounced tendency toward total laryngectomy even in cases in which thyrotomy would seem adequate.

Dr. Henry L. Swain, of New Haven: I would ask a question in regard to the technique. Have you used adrenalin in these cases? If not, is there any reason against its employment?

Dr. H. W. Loeb, of St. Louis: I have one case of laryngec-

tomy for cancer which is living ten (10) years after the operation, and one case of thyrotomy living three (3) years after the operation.

Dr. D. Crosby Greene, Jr., of Boston, closing the discussion: With regard to the question of anaesthetic, I am not sure that I made plain my method. I do use a general anaesthetic. I have the patient placed profoundly under the influence of ether, preceding this with a hypodermic injection of morphine and atrophine. *The ether is not removed until the stage of the operation is reached in which the larynx is opened.* As a rule, the patient comes out of the ether as he is returned to bed. The Trendelenburg position is maintained during the operation.

With regard to the location of the growth as influencing the type of operation, it seems to me the important point is not so much whether the growth is anterior or posterior, but whether it can be removed with a wide margin of tissue. It is not so much the location as its accessibility and the avoidance of injury to the growth in its removal. This is the important point.

With regard to Dr. Coakley's remarks about sewing up the cartilage, I do not suture the cartilages together; they are brought together and the overlying soft tissues sutured, excepting at the lower end of the wound, which is left open for drainage.

With regard to the use of adrenalin, it does help in defining the limits of the growth. But when closing the larynx up immediately after the operation there should be no subsequent oozing. If adrenalin has been used there is liable to be a good deal of oozing after its effect has worn off. In the employment of the method described, there practically is no bleeding in the larynx at all until after the sub-perichondrial elevation has been accomplished. The removal of the growth can then be accomplished quickly and accurately.

As to Dr. Richards' case with cachexia, I cannot account for it. Probably there was some other source of the cachexia than the cancer. It is a rather unusual case.

I have had one case of stenosis where I had to put in a tracheal tube for stenosis after operation and the patient is wearing the tube at the present time.

It is my custom to open the larynx without doing a preliminary tracheotomy, and close the wound after the operation.

COMMINUTED FRACTURE OF THE LARYNX—ACCIDENTAL TRACHEOTOMY—MULTIPLE TRAUMA—EXTENSIVE FROSTBITE—RECOVERY.

BY D. BRYSON DELAVAN, M.D.

The subject of this history was formerly a well-known figure in the leading throat clinics of Central Europe. While his case was more than once exploited in the public press, it does not seem that it has ever appeared in the medical literature of this country. My own experience, both in general surgery and in that of our own special department, has not furnished a parallel to it, and I am now placing it upon record that it may not be lost. About twenty years ago the patient came to the United States with the intention of making a living by the exhibition of his throat at various medical schools and by any other means within his power.

He was indeed a forlorn spectacle, that poor Roumanian Jew, who, with the help of two canes, hobbled painfully into our lecture room at the College of Physicians and Surgeons one day, offering his throat for examination and presenting to us the story of his incredible sufferings. The haggard expression of his scarred countenance and the peddler's tray with its meagre outlay of simple wares gave evidence of dire suffering manfully met. He was wearing a tracheotomy tube, and came to us for the purpose of demonstrating the introduction into a stenosed larynx of the dilator of Professor Schrotter, of Vienna. From Schrotter, as well as from Professor Stoerck and others of Vienna, from Professor Fraenkel, of Berlin, Professor Moritz Schmidt, of Frankfurt, and several other eminent European laryngologists, he brought strong letters in which the writers said that he had been in the habit of exhibiting himself at their clinics and vouched for the truthfulness of his story. They bespoke for him the kind consideration and generosity of their colleagues in other parts of the world. As to these letters, I was familiar with

the handwriting of many of their authors. They were beyond question genuine. Moreover, I have heard by word of mouth from several of the writers the details of this most remarkable case, although no further corroboration was necessary than that presented by the scars resulting from the injuries claimed to have been received.

The history is one of unique interest, not only by reason of the extraordinary vitality shown in the face of a series of injuries any one of which might easily have proved fatal, but because of the almost miraculous sequence of incidents, each instantly essential to the immediate saving of the man's life. The patient, Ivan ——, had been a prosperous young citizen of a town in Eastern Roumania which lay in the midst of a country * * * sections of which were wild and badly protected. It was New Year's time, and the tax gatherer of the district had completed his collections and, with the revenue money in his possession, attended a banquet and dance upon New Year's Eve at the fine residence of a wealthy land holder. At the close of the festivities, shortly after midnight, the tax gatherer with his treasure and a party of seven companions, including the subject of this history, departed for their town, about sixteen miles distant, in a large three-horse sleigh. The ground was thickly covered with snow, the sky brilliant with stars and the temperature below zero. Some of the journey lay through a particularly wild and desolate region, but the party was in high spirits and apparently not suspicious of danger. The fact that they were conveying a considerable sum of money had become known, however, and in the loneliest part of the way they were attacked by a band of outlaws. The place chosen for the ambush was at the edge of a dense wood not far from the banks of a stream. The suddenness of the onslaught made effective resistance impossible and all the party were quickly massacred, all, at least, except Ivan ——, who succeeded in concealing himself for a time under the fur robes in the bottom of the sleigh. His hiding place was soon discovered and he was dragged out and stripped of his valuables and clothing. A rope was then fastened around his neck and he was hanged from the limb of a neighboring tree.

The attention of the bandits was then diverted from him in their eagerness to complete the robbery of the other victims. Left to himself and thus suspended in the air, his struggles were so desperate that he not only created a multiple fracture of his larynx, but also succeeded in breaking the rope. He fell to the ground rapidly suffocating, but nevertheless able to crawl into the bushes nearby, where he hoped to find concealment. His loudly stertorous breathing, however, quickly revealed his retreat. Two of the ruffians rushed upon him, one with a large knife, the other with a revolver. The one armed with the knife, evidently approaching in the direction of the victim's head and at the left side, inflicted upon him three vicious cuts. The first extended from the margin of the right jaw upward across the face and over the forehead, passing completely through the cheek and partly dividing the upper eyelid, but fortunately escaping the eye itself. The second attack resulted in a transverse incision straight across the throat, passing directly between the thyroid and cricoid cartilages and dividing the crico-thyroid membrane. It failed to injure, however, either the carotids or the jugulars. Through this opening, as the man's head fell backwards, air was instantly admitted to the lungs and the frightful dyspnœa occasioned by the fracture and resulting collapse of the larynx was overcome. The third slash, diagonally across the abdomen upon the right side, completely opened the peritoneal cavity throughout nearly its entire extent. Through this wound the intestines at once protruded, although apparently they had not been incised. To make sure work of their victim, the revolver was then emptied into him. Meanwhile the bodies of the other victims had been carried to the bluff at the bank of the stream, there about twenty feet above its surface, and had been thrown one by one down upon the snow covered ice below. Ivan, apparently dead, was among the last to be so disposed of. The band then departed with their rich booty.

Some time during the next morning several peasants happening to come that way saw evidences of the tragedy that had taken place and, following the bloody trail from the woods

to the river, discovered the bodies of the victims below. With all speed they summoned help. Examination showed that all of the party were dead excepting Ivan. In him there seemed to remain a faint spark of life, but in addition to his other injuries his hands, feet and ears were badly frozen. Doubtless, nevertheless, the effect of the intense cold had been to stop the hemorrhage from his fearful wounds. He was taken to the nearest hospital, where his injuries were successfully treated, the wound in the neck closed and a tube inserted through the laryngeal opening. The effect of the frost bites, however, was very severe, so much so that the patient was obliged to undergo a Symes amputation of the right foot, a partial amputation of the left foot, and the loss of most of the fingers of both hands. Strangely enough, notwithstanding all these injuries, he recovered and, after wandering about the clinics of Europe for five or six years, came to the United States.

Incredible as this history seemed, the best evidence of its truthfulness existed in the scarred and mutilated body of the man himself, for there was not one of the wounds alluded to that was not represented by its own terrific scar or by the loss of an important member. Laryngoscopic examination proved the serious nature of the injury inflicted upon the larnyx, for there had been a multiple fracture of the thyroid cartilage, fracture of the cricoid, fracture of the hyoid bone and such collapse of the interior of the larynx that the resulting stenosis had been impossible of relief by the then existing methods of treatment. The permanent wearing of a tracheal canula was a necessity.

The sequence of events by which the man's life was saved are worthy of note.

Had he not slipped under the robes to the bottom of the sleigh he would have been massacred with his companions at the first onslaught. His struggles while hanging, although they fractured the larynx, released him from the rope. When he hid in the bushes certain to die from dyspnoea unless relieved, the stridor of his breathing revealed the place of his concealment and called forth a murderous gash which provi-

dentially opened the one place through which the desperate need of air could be supplied. The pistol shots while penetrating the skin probably did not enter deeply into the body. At all events no important organs seem to have been injured by them. From the nature of the principal wounds the man should have been quickly exsanguinated, but fright and shock must have produced nature's anesthetic, syncope, and with syncope, together with the intense cold of the occasion, pain was overcome and hemorrhage controlled. A sheer fall of twenty feet upon hard ice would have been enough to kill. But Ivan's fall was broken by the presence of the snow and of the bodies of his comrades, the latter possibly furnishing sufficient warmth to keep him alive. The purity of the cold winter atmosphere saved his wounds from infection and the rest was accomplished by what appears to have been remarkably skillful surgical care. The history of the injuries to the larynx and their results are particularly instructive as far as the prevention of stenosis is concerned. The treatment by means of Schrotter's dilators, the best at that time known, was a complete failure. The employment of modified O'Dwyer tubes, after the method of Dr. John Rogers, Jr., of New York, especially if applied promptly after the occurrence of the injury, would have saved the patient a lifetime of disability and suffering.

The final history of this case is even more tragic than what has already been related. He became successful in a modest way as a peddler, and in the course of two or three years proposed and was accepted in marriage by a young woman in New Jersey. A few years afterwards, in reading the morning paper, I saw a statement to the effect that what the robbers of his native country had failed in doing had been successfully accomplished by his brother-in-law, who, wearying of his presence, had shot him dead.

REPORT OF A CASE OF ULCERATION OF THE LARYNX, PERICHONDRITIS OF THE ARYTENOID CARTILAGES, ABSCESS AND PARTIAL EXFOLIATION OF BOTH CARTILAGES, RESULTING FROM TYPHOID FEVER.

BY J. H. BRYAN, M.D.

This case is reported in order to emphasize the importance of making regular and systematic examinations of the upper air passages, especially of the larynx, in all cases of typhoid fever, in order to detect the early changes that take place in the mucous membranes of the upper air passages in this disease.

On the night of October 12, 1912, I was called to see Major C., U. S. A., aged 40, at the Walter Reed Hospital, suffering from a severe case of typhoid fever, then in about the fiftieth day of the disease. I was called to see the patient because of his difficulty in breathing and loss of voice. The following history was given me by the attending surgeon: In September this officer returned to his duties in Washington, after a Western trip, suffering from typhoid fever. The disease ran rather a severe course from the beginning, with high temperature and several severe intestinal hemorrhages. The temperature gradually subsided, and within a week or ten days of my seeing the case he was supposed to be convalescing. About four days before I was called to see him the patient was thought to have taken cold, a slight laryngitis developed, accompanied by a cough and a husky voice, but at no time did he complain of any pain in the region of the larynx.

When I saw the patient he presented a very emaciated appearance, with an anxious expression of the face, skin bathed with sweat, respiration rapid and shallow, temperature 100, pulse small, thready and rapid, and voice husky. Laryngoscopic examination showed fauces very much congested, but no ul-

ceration in this region. There was extensive swelling of the right ventricular band, which overlapped and partially obscured the corresponding true vocal cord which is in the median line and did not move on deep inspiration; there was also seen a deep ulceration on the under surface of the ventricular band, which seemed to extend to the free margin of the true vocal cord. Both arytenoid cartilages were markedly swollen and posteriorly were bathed in a thin, grayish secretion, probably purulent in character. The left vocal cord moved somewhat on inspiration, leaving a narrowed glottis, sufficient, however, to admit of respiration.

The gravity of the situation was explained to the family and a tracheotomy advised, but owing to the fact that he seemed to be breathing deeper and was more quiet, I left him about midnight, cautioning the surgeon in charge to do an immediate tracheotomy if his respiratory difficulty increased. I saw the case again the next morning at 10 o'clock, and while the patient had passed a restless night, he seemed, on the whole, somewhat improved as to his local condition, but not so well generally. The examination of his larynx showed the condition about the same as when last seen at midnight, with the exception that the excursion of the left vocal cord was greater, giving a much wider chink to the glottis and thereby admitting of freer respiration. The consent of the family having been obtained, it was arranged that I should return to the hospital at 2 o'clock, when a tracheotomy would be done. I had not been away from the hospital more than an hour when the patient had a severe coughing spell, which brought about a spasm of the larynx, necessitating an immediate tracheotomy, which was done by Captain Smith, the medical officer in charge of the case. The patient did not breathe after the trachea was opened, and died almost immediately, although stimulation and artificial respiration were resorted to. Owing to the prolonged illness the patient's heart was greatly weakened, and his death was due to heart failure about the time the trachea was opened. The larynx was removed, and unfortunately placed in Muller's fluid, which discolored the specimen.

The following day Major Russell, of the Army Medical

School, and I opened the larynx, and found the following conditions:

The right ventricular band swollen and a large ulceration on its under surface; the true vocal cord ulcerated on its free margin; both arytenoid cartilages completely detached and lying loose in their capsules, the right cartilage being held in

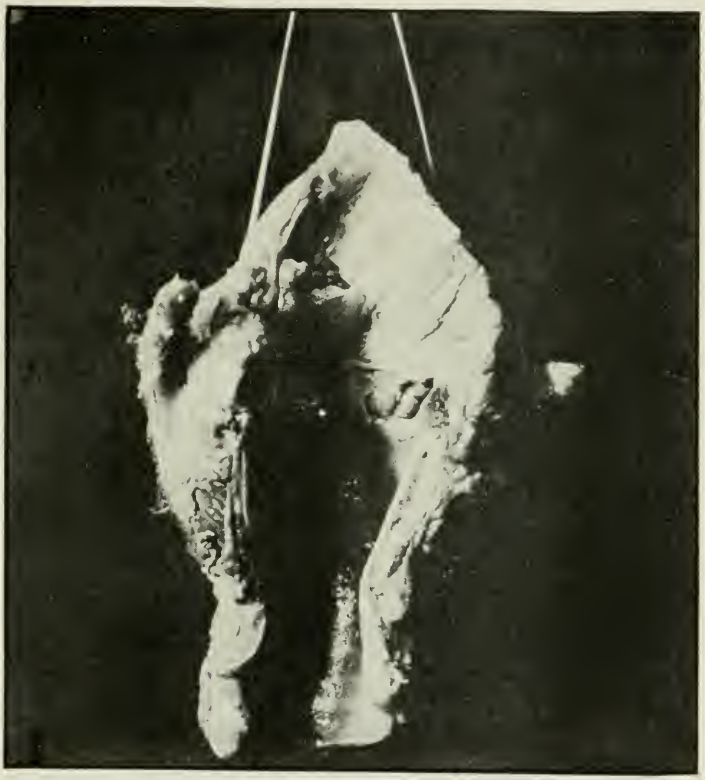

Fig. I.

position by only a small membranous attachment. A bacteriological examination made by Major Russell proved negative.

The photograph (Fig.I.) of the specimen does not show the destructive changes that were found in the larynx as clearly as the natural specimen; the shadow is so deep that the right arytenoid cartilage is with some difficulty seen, but it is suffi-

ciently distinct to enable us to see the destructive changes that have taken place. Fig. II. shows conditions somewhat more clearly.

In the light of what has been accomplished by anti-typhoid vaccination in the United States Army, it may be asked why this officer had not been immunized. Several years ago he

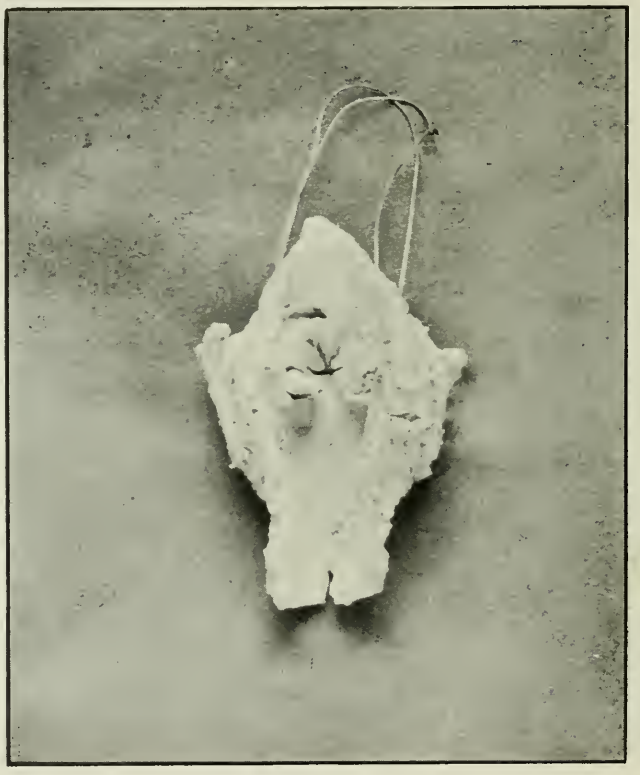

Fig. II.

contracted pulmonary tuberculosis, and for a while was under treatment for this disease at Fort Bayard, but later was discharged cured, and restored to duty in Washington. In view of his previous tubercular history, and as tuberculosis is a well recognized contra indication to typhoid vaccination, it was deemed unwise to subject him to this treatment.

This brings up a very interesting point in connection with typhoid prophylaxis, and that is, that typhoid vaccine acts very much in the same way as tuberculin, with a constitutional reaction and fever lasting several days. Some French surgeons claim to have had cases in which latent tubercular lesions had been excited and made quite evident after typhoid vaccination.

Owing to the insidious character of the development of this complication, it is wise that a laryngeal examination should be frequently made of typhoid cases, especially so when there is the slightest change noted in the character of the voice, cough, or difficulty in respiration. Jackson[1], in a very interesting article on this subject, after a study of 360 cases, quotes Dr. John W. Boyce on this point, who says it is a question of great interest whether it is possible for these affections to cause a fatal termination without such symptoms as shall plainly indicate the nature of the occurrence. He further says, theoretically it seems impossible that a man should choke to death without the conspicuous symptoms of asphyxia, particularly cyanosis, but observation has convinced him that it is only too true; that these cases may sink away as peacefully as a baby goes to sleep, and that one may not in the least suspect what is happening.

Jackson divided his cases into sub-acute catarrhal laryngitis, ulcerative laryngitis, and perichondritis. The first was found to be present in 227 cases (or 63 per cent), the second in 68, and the third in 17, of the 360 cases.

St. Clair Thomson[2], in the chapter on laryngeal complications in typhoid fever, in his work on Diseases of the Nose and Throat, gives the frequency of occurrence from the European standpoint as follows:

Landgraf, 11 per cent of all fatal cases.
Griesinger, 26 per cent of all fatal cases.
Kanthack, 26 per cent of all fatal cases.
Ouskow, 30 per cent of all fatal cases.

It is difficult to arrive at any conclusion as regards the com-

[1] Jackson, Journal American Med. Sciences, Nov., 1905, page 845.
[2] St. Clair Thomson, Diseases of the Nose and Throat, page 671.

parative frequency of this complication of typhoid fever in this country and abroad. The figures given by Jackson seem to show that a much larger number of cases of laryngeal involvement occur in this country than is indicated by the figures given by Thomson. The epidemic in which Jackson made his observations, however, was an unusually severe one, and his subjects were largely of a poorly nourished type, and this may account for the apparently greater frequency of this complication in this country.

We will not be able to get at the truth of this matter until more careful observations are made, not only in the hospitals, but in private practice as well.

Discussion.

Dr. Emil Mayer, of New York: A very important thing to bear in mind, no matter how great the perichondritis is, how will the larynx appear subsequent to the treatment and are good results obtained. Twelve years ago I was called in to see a post-typhoid perichondritis case, and had to perform tracheotomy in a great hurry. The subsequent history has previously been presented. Intubation was performed, and eventually the tube was removed. There has never been any difficulty in breathing. There is still a decided thickening over the arytenoid, and the results are encouraging. We should not lose hope no matter how bad the appearances in the larynx.

Dr. Chevalier Jackson, of Pittsburgh: I wish to emphasize a number of points which have been very clearly brought out by Dr. Bryan. It is true that in the Pittsburgh epidemic, in which the observations referred to were made, the disease was exceedingly virulent. In one hospital there were 700 cases of typhoid fever in a single year, and almost all of them of very severe type. All forms of complications were very common and the larynx was quite often involved. Now we rarely see a case of typhoid fever in Pittsburgh, and still more rarely is there any laryngeal complication. Nevertheless, as Dr. Bryan points out, the larynx of every typhoid fever patient should be watched, because, while it is true that the symptoms calling attention to the larynx may be so late that it has become common to call the laryngeal complications "post-typhoid," the laryngeal trouble starts in the second or third week of the disease. By the time chondrial necrosis and

abscess has resulted the patient is convalescent of his typhoid fever proper. Dr. Boyce, who worked with me from the internist point of view, has said that the fact that a typhoid fever patient with laryngeal complications can choke to death calmly and peaceably has led to typhoid laryngeal cases being labeled, sudden heart failure in convalescence. Recent observation leads one to believe that the same thing occurs in pneumonia, scarlet fever, and probably other diseases. In many cases the respirations are rapid and shallow, pulse small and thready, which do not suggest obstructive dyspnea. One point I have discovered in my work is that the destruction in the larynx is due to the mixed infections, and that oral sepsis is the most important factor in the development of severe cases. For prophylaxis the thorough cleansing of the teeth and mouth with frequent rinsings of diluted alcohol, about one part to five, is most important. For treatment there is one remedy that will cure every case; namely, tracheotomy. This is entirely apart from stenosis. Regardless of the presence or absence of dyspnea, all these cases should be tracheotomized as a therapeutic measure. Doubtless, as suggested by Dr. Bryan, vaccine prophylaxis of typhoid fever will lessen the frequency. The specimen is a very remarkable one, and Dr. Bryan's accurate observation of the lesions present during life is an exceedingly valuable contribution to our knowledge of the subject.

Dr. Henry L. Wagner, of San Francisco: Some years ago I had the opportunity of examining the diseased larynx of typhoid fever patients; they could be classed into two groups: (a) specific typhoid ulceration, and (b) ulceration due to decubitus. In the latter cases no typhoid bacilli were found at times, though in large numbers the staphylococci and streptococci—so frequently found in the mouth.

The prognosis is better in the former group than in the latter.

I believe in the constant use of oral antiseptics in typhoid fever.

Dr. Burt R. Shurly, of Detroit: Are any changes found in the lungs?

Dr. Wagner: No.

Dr. Albert C. Getchell, of Worcester: I will report two cases. In one there was a definite lesion in the larynx; tracheotomy was performed and death followed. In the other case the lesions in the larynx caused obstructive breathing, but this patient recovered. I do not recall whether the typhoid fever in these cases was very severe or not.

Dr. Henry L. Swain, of New Haven: I had a case that closely resembled the one related by Dr. Bryan. The symptoms which were originally called to my attention were those referable to a paralysis of the posticus muscles. Tracheotomy had to be quickly performed, but subsequently the patient died because of a hemorrhage into the trachea. At autopsy the laryngeal condition was found to be very similar to that described by Dr. Bryan, as there was complete detachment of the cricoid in the pus pocket in the larynx wall. Death in this instance occurred some ten days or two weeks after the original tracheotomy.

Dr. Joseph H. Bryan, of Washington: After tracheotomy in these cases no relief is obtained, except so far as breathing is concerned.

DECANULATION AND EXTUBATION AFTER TRACHEOTOMY AND INTUBATION RESPECTIVELY.

BY CHEVALIER JACKSON, M.D.

The different forms of laryngeal stenosis associated with difficult decanulation or extubation may be classified into the following types:

1. Panic.
2. Spasmodic.
3. Paralytic.
4. Ankylotic (arytenoid).
5. Neoplastic.
6. Hyperplastic.
7. Cicatricial
   - (a) Loss of cartilage.
   - (b) Loss of muscular tissue.
   - (c) Fibrous.

Panic. Breathing through the neck with a properly placed tracheotomy canula is so much easier than breathing through the mouth that once the patient becomes accustomed to it, for quite a while he does not feel that he is getting enough air through the mouth, even though the larynx is perfectly patulous. In addition to this there is a "nerve cell habit" arising from previous experience with the stenosis that terrorizes the patient the moment he feels the slightest dyspnea. In children crying tends to increase stenosis (by disturbance of respiratory rhythm and venous engorgement), and fright is very apt to do so in either adults or children. Glottic spasm may or may not contribute. All these things taken together may be called "panic" and constitute quite a formidable obstacle to decanulation where there is no real stenosis of the larynx.

Spasm. Spasmodic stenosis may be associated with panic, or may be excited by subglottic inflammation. It is usually overcome by the same means as those suggested for panic, together with the treatment of the inflammatory condition that

may be present. Doubtless one of the chief causes of adductor spasm is the prolonged wearing of an intubation tube, especially a large one, which prevents activity of the adductors, and of the abductors, for the action of these two sets of muscles is reciprocal, and the normal balance is, of course, interfered

Fig. 1. From a photograph of a patient taken two years after cure of obstinate cicatricial post-typhoid laryngo-tracheal stenosis, by laryngostomy.

with by the presence of an intubation tube for a long period. Three methods of treatment may be used in these cases to get the patient permanently extubated. 1. Replacing the intubation tube with a special one, which has a very narrow neck with a long anteroposterior lumen in order to allow free glot-

tic action for a time, until muscle balance is restored. 2. In a few cases of not very severe type it is possible to get them well by a patient extubation with replacement as soon as the child begins to get blue. This requires a facile intubator who has plenty of confidence in his ability to slip in the intubation tube promptly and without trauma. This method will not succeed in a violently spasmodic type, where the symptoms are so urgent and severe that the tube can only be left out for a

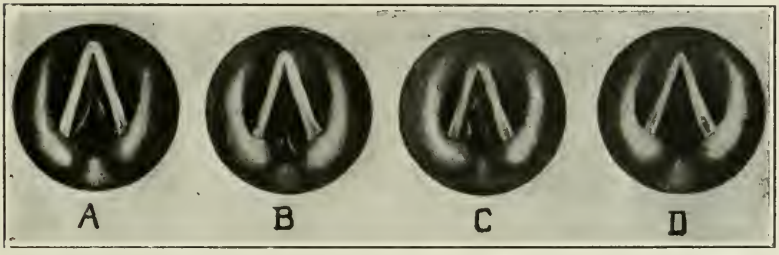

Fig. 2. Illustration of the effectiveness of galvano-cauterization of post-diphtheritic subglottic hypertrophic stenosis. The views represent the larynx as seen through the direct laryngoscope which hides the epiglottis. A shows the larynx immediately after the removal of the intubation tube. B. Five minutes later. The hypertrophic subglottic masses on each side are seen to have closed in like intumescent turbinals. C. The left mass has been cauterized and is bound down by a linear cicatrix parallel with the long axis of the trachea. D shows the larynx after cure by repeated cauterizations.

few seconds. But in those cases that require a half hour or more to close it is quite often successful. 3. Tracheotomy for extubation is the quickest method of cure in purely spasmodic cases without organic stenosis. The wearing a tracheotomy canula for a week or two will permit the restoration of muscle balance, and by corking the canula as elsewhere herein mentioned, the child can be gradually weaned away from the canula, and thus permanently extubated and decanulated.

Paralysis. Bilateral laryngeal paralysis causes a severe stenosis of the larynx, provided the paralysis is not cadaveric. In cadaveric paralysis there is usually sufficient breathing space and this has led to nerve division to relieve stenosis. I agree with Charles H. Knight that nerve division has been a failure, but I have thought that nerve excision might have

yielded better results. In decanulation for paralytic laryngeal stenosis three methods of treatment may be followed. Cordectemy has yielded good results in rare instances, the cords being excised either by thyrotomy or endolaryngeally. The author had one success after evisceration of the larynx endoscopically by the direct method. The results of thyrotomic evisceration are absolutely ideal in cases where there are no lesions other than the bilateral paralysis. Formerly, when it was thought that excision of the cords meant permanent loss of voice one might hesitate to recommend evisceration. In two cases operated upon by the author a fairly loud, though very rough phonation, mostly in a monotone, was obtained by both the patients. It was a good useful voice, though, of course, it did not have the flexibility that we see after thyrotomy, for conditions in which there is unimpaired mobility of the arytenoid joints. In both of these cases, however, I did a mucoperichondrial dissection, taking out all the soft tissues and not simply the cords alone. Both patients were permanently decanulated. Great care is necessary to make sure that all of the sub-glottic tissue is dissected out. Of course the operation is only to be recommended when the paralysis is unassociated with essentially fatal conditions, such as aneurysm and malignant mediastinal tumors. George L. Richards reports a case in which recovery from a laryngeal paralysis occurred, but that was in a child. As a rule, it may be said that when bilateral paralysis remains twelve months it will never be followed by recovery, because of muscle atrophy or joint stiffening. Therefore the operation is justifiable, if the patient wishes to be decanulated and considers some impairment of voice more than outbalanced by the getting rid of the canula and being made absolutely safe from asphyxia. It is rather appalling to note the number of patients with bilateral laryngeal paralysis that have died as a result of accident to the intubation tube or canula. In bilateral recurrent paralysis of recent occurrence it may be worth while to attempt suturing the recurrent to the pneumogastric, provided the paralysis is recent, even if it is not peripheral. Monolateral paralysis does not usually cause sufficient dyspnea

to call for a tracheotomy, but it is worthy of note that excision of a scar and suture of the recurrent laryngeal after injury has been successful in restoring motion to one cord (J. Shelton Horsley and Clifton M. Miller).

Ankylosis. Bilateral ankylotic conditions of the crico-arytenoid joints may prevent decanulation until the laryngeal stenosis is relieved. In one case of this kind, evisceration of the larynx, as mentioned above for paralysis, completely cured the stenosis in a man forty years of age. Of course, this operation is not to be advised, except in such cases as have remained regid for a period of twelve months or more, and it is not meant to include the fixation that is associated with malignant, tuberculous or luetic infiltrations.

Neoplasms. Decanulation in neoplastic cases will, of course, depend, so far as stenosis is concerned, upon the nature of the growth and its curability. In malignant conditions after thyrotomy, stenosis practically never occurs. After hemilaryngectomy, stenosis may follow from a flaccid condition of the side wall of the larynx, or there may be a cicatricial contraction diminishing the stenosis. As a rule, in these cases I believe the best treatment is continuous dilatation from the prolonged wearing of a large intubation tube, though it is better to defer intubational dilatation until quite certain that the malignancy is not going to recur; because if the malignancy recurs re-operation for malignancy can be so planned as to take care of the stenosis, by evisceration of the normal half of the larynx. In cases of stenosis associated with benign tumors other than papillomata, decanulation rarely presents difficulties. The removal of the tumor usually restores the laryngeal lumen. (Case 3.)

Papilloma. Papilloma presents quite a different problem, inasmuch as the growths persistently recur, though, of course, unlike malignancy, they do not infiltrate. Their removal usually restores the lumen and the patient is thus readily decanulated; but recurrence must be carefully watched for and removed before the stenotic stage is reached. As a rule, papillomata should be removed without doing a tracheotomy, unless the circumstances are such that the child cannot be watched

carefully after removal, in which case tracheotomy may be required to make the child safe over night; and, certainly, in any case of doubt, tracheotomy ought to be done any way, in order to be on the safe side. Unfortunately, unless very carefully watched, the child is not absolutely safe even with the canula, because the canula may come out, or papillomata may encrouch upon the inner orifice of the tube and a portion of tissue may be detached and occlude the tube completely. There have been quite a number of such cases where the child has been found dead in bed. It has happened in three instances in which the attending physician had arranged with Dr. Patterson or me to take care of the child, and later the report came of its having been found dead in bed from occlusion of the canula during sleep. In cases in which a tracheotomy has been done and the papillomata have afterward been removed, the question arises whether or not the canula should be abandoned. Where the child is in the hands of a nurse skilled in tracheal work, it, in most instances, is as well to remove the canula after the larynx is healed and it is found that the growths are completely removed. When this is done, however, it is absolutely necessary that the larynx be closely watched in order that the removal of recurrences can be undertaken promptly, for it is one of the peculiarities of papillomata that the dyspneic symptoms often set in extremely suddenly, and it is a great mistake to wait for the onset of dyspnea to have the child return for treatment. Removal should be repeated as often as any point of recurrence becomes visible. If the circumstances are such that this can be done, it is better to abandon the canula early in most instances, because in my experience the wearing of a canula, far from causing the disappearance of papillomata, really causes their extension downward toward the tracheal wound, possibly because of lessened aeration above the canula, and in many instances the entire subglottic trachea will become packed full of papillomatous tissue, especially if the tracheotomic canula is left long *in situ* without frequent cleansings. If papillomata have been carefully removed, and no injury has been done to the motor area of the larynx, there will be no cicatricial stenosis. Quite a number of cases, however, come in where indirect

methods have removed masses of normal tissue, which has been followed by severe cicatricial stenosis, or, by operations by either the direct or indirect methods, the motor area may be damaged so as to lead to ankylotic stenosis. Dr. Harmon Smith is obtaining excellent results from fulguration in the treatment of papillomata in children, and this seems an ideal method, as it leaves no vicious cicatrices. Dr. Clifton Miller and Dr. A. L. Gray are using the Roentgen ray externally. I am at present using the ray in conjunction with laryngostomy in one case.

Compression. Peritracheal neoplasms occasionally cause compression stenosis, as do also hypertrophy of the thymus and thyroid. Decanulation in a thymic compression case is very readily accomplished by either thymopexy or a partial thymectomy. A struma can be dealt with by the usual well known methods.

The hyperplastic and cicatricial types of organic stenoses preventing decanulation may be classified etiologically as follows:

1. Tuberculosis.
2. Lues.
3. Scleroma.
4. Acute infectious diseases.
   - (a) Diphtheria.
   - (b) Typhoid Fever.
   - (c) Scarlatina.
   - (d) Measles.
   - (e) Whooping Cough.
5. Decubitus.
   - (a) Canular.
   - (b) Tubal.
6. Trauma,
   - (a) Tracheotomic.
   - (b) Intubational.
   - (c) Operative.
   - (d) Suicidal and homicidal.
   - (e) Accidental (by foreign bodies, bullets, etc.)

Most of the organic conditions, outside of the paralytic and neoplastic forms, are almost all the result of inflammation, often with ulceration and the secondary tissue changes. In the infective granulomata, such as lues and tuberculosis, and

in the acute infectious diseases, it is practically always the mixed infections from oral sepsis running riot that do the harm. The chief exception to this is diphtheria, which in many cases is distinctly a necrotic process, wherein the replacement of the lost tissue by cicatricial tissue causes the stenosis either by cicatricial contraction or by the bulk of the newly formed inflammatory infiltrate or of pus collections.

Tuberculosis. In the rare cases in which laryngeal tuberculosis of such severe type as to demand tracheotomy is cured decanulation usually presents little difficulty after the infiltrations are reduced. The reduction of these infiltrations by the galvano-cautery through the laryngeal speculum is readily accomplished. Should cicatricial stenosis from ulceration remain, it is, of course, to be treated in the same way as cicatrices in other cases, by laryngostomy. The author has seen but a single case of this kind. In the non-cicatricial forms, which are relatively common, laryngostomy is not necessary, and direct application of the cautery will give such a degree of reduction of infiltration as to give an ample lumen.

Lues. Swain reports a case of luetic immobility of both cords in which the wearing of an intubation tube intermittently gained sufficient lumen in the larynx for respiration until general medication cured the patient. Under the careful watchfulness of Dr. Swain, such a procedure was safe, but as a rule patients are far safer with a tracheotomy, though decanulation may be somewhat more difficult than getting rid of an intubation tube. Luetic cicatrices are proverbally prone to return, and are particularly vicious in contraction. Prolonged stretching with oversized intubation tubes following either incision with the galvano-cautery or excision with cutting forceps is sometimes successful, but usually laryngostomy is required. In those old cases of chronic luetic fibrosis, which are, in a sense, paraluetic conditions, little, if at all, amenable to the older methods of medication, salvarsan has accomplished wonders. It has even been claimed that cicatrices of luetic origin have been benefited. It would seem, however, that in such cases there must have been an underlying fibrosis

of the nature of a luetic lesion, and not purely and simply a cicatrical condition following such a lesion. Scar tissue is scar tissue, regardless of what produced it, and we must rely upon laryngostomy for the cure of most of these conditions.

Scleroma. Dr. Emil Mayer recommends the use of radiotherapy in the treatment of scleroma. If the stenosis is severe, doubtless it would be well to open the larynx externally and keep it open as in laryngostomy, so that the applications of the ray could be direct to the scleromatous tissue. So far the results of treatment of scleroma have been unsatisfactory and they probably constitute the only cases in which decanulation is impossible.

Diphtheria. Diphtheritic cases may be of the panic, spasmodic or rarely the paralytic types; but more often the stenosis is of either the hypertrophic or cicatricial forms. After intubation, especially if prolonged, there may be a hypertrophic condition, which is manifest in two ways: 1. An edematous condition of the upper orifice of the larynx, usually worse anteriorly around the base of the epiglottis, but also in some instances extending backward over the glossoepiglottic fold and the ventricular band, either or both. (Fig. 4, Case 4.) To this form I have given the name supraglottic hypertrophy. It resembles somewhat the ordinary acute laryngeal edema, except that it seems to be chiefly anteriorly and is more sharply limited than the latter lesion usually is, and it has somewhat of a tendency to overhang and occlude inspiration more than expiration. The supraglottic hypertrophy I have found, in some instances, to be due to the wearing of an intubation tube which has a sharp angle at its upper anterior edge. In one instance, the tube was smooth and rounded in this position, but was entirely too thick in the neck for the age of the child. Where these or any other defects in the tube are suspected of being responsible for the trouble, it is wise to change the tube for one of correct model and size and await results before attempting any more radical treatment, though local applications of argyrol can be made while testing out the effect of a correct tube, though excessive polypoid surpraglottic hypertrophy should be excised. The infraglottic type is usually

bilateral. I have many times watched these cases in the direct speculum, and this is what happened: After the removal of the intubation tube the laryngeal lumen appeared large and ample, the cords pink, and there seemed to be an ample lumen below. But within a few minutes, or more, in some instances, the subglottic tissues gradually began to close in from each side in the form of two lateral masses rather pale in color, usually, though in some cases quite red. As the masses gradually encroached upon the lumen dyspnea began to set in, and it was usually as much expiratory as inspiratory, though in some instances the reverse was the case, why, I do not know, unless it is that the child instinctively draws its breath in quickly as compared to the duration of the expiratory movement. Either the supraglottic or infraglottic forms may be treated by tracheotomy, and in fact they are very much safer with a tracheotomic canula in place than intubated. In most instances, however, the tracheotomy will not cure. In the supraglottic type the edematous tissue may have to be removed surgically, which is readily done through a direct speculum. Occasionally applications of argyrol 10 per cent. carefully painted by the direct method will hasten the reduction of the redundancy. In the infraglottic type of hypertrophy, tracheotomy will only occasionally relieve the condition and resort must be had to pressure dilation with the author's divulser repeated just as often, as possible without causing undue reaction. In this divulsion, care should be taken not to cause any traumatism, and especially in the neighborhood of the motor area. The dilatation should be subglottic, and with care there is no need of injuring any of the muscles of either of the cricoarytenoid arytenoid joints. Intractable forms which failed to yield to tracheotomy or divulsion may be cured by vertical linear applications of the galvano-cautery. Cases in which this fails will require laryngostomy, which I believe will cure every case, and the duration of laryngostomy, under these circumstances, is very much shorter than in the cicatricial type. In the cicatricial type the fibrous tissue may

take many forms. In some cases there is a band running across the larynx from one side to the other; it may be between the two ventricular bands, between the two cords, or from one band to the opposite cord, or to the same cord of the same side. Occasionally the cicatrix is in the form of a funnel with a minute opening at the bottom of the funnel. In some instances there is a web anteriorly, which very much diminishes the air space and may interfere with phonation or may not, depending on the degree of approximation possible. In two instances I have seen a cicatricial mass between the arytenoids posteriorly with ankylosis of both joints, leaving only a very small opening close to the anterior commissure. This is unusual. More frequently the opening is somewhere in the posterior two-thirds. The management of the panic and spasmodic types of post-diptheritic stenosis has been previously herein considered.

Typhoid Fever. About ten years ago, when typhoid fever was prevalent in Pittsburgh, I made an investigation of the laryngeal complications (Am. Journal Med. Sciences, Nov., 1905), with the aid of Dr. Ralph Duffy and Dr. Joseph H. Barach. It was found that the ulcerative lesions in the larynx were practically always the result of a mixed infection, and in some instances they were due to thrombosis of a small vessel with subsequent necrosis. When the ulcerative processes reached the perichondrium cicatricial stenosis was almost certain to follow, and practically all of the cases with chronditis (once of one artyenoid preventing formation of an adventitious band) required tracheotomy for acute edematous stenosis. The after treatment of these cases was chiefly by prolonged intubation, with special intubation tubes, the author's T-shaped canula, and in some instances laryngostomy. The results have been previously reported.

Scarlatina may be followed by acute laryngeal stenosis, due to infection with either strepococcic or other pyogenic organisms There may be cellulitus of the neck, chondritus and necrosis, but these are rare. In any event, the stenosis following is cicatricial and is handled just as any other cicatricial stenoses. Influenzal chronditis produced a tracheal stenosis in one of my cases.

Trauma. Occasionally foreign bodies, by a prolonged sojourn may ulcerate through from the esophagus into the trachea. The author has reported two such cases. In other instances prolonged sojourn of a foreign body in the larynx has been known to cause stenosis. Dr. Joseph A. White reports an exceedingly interesting case of laryngo-esophageal fistula following perichondritis and abscess due, probably, to the trauma of a foreign body, and subsequent infections. Dr. White's skillful handling of the subsequent stenosis illustrates the great difficulties to be met with in certain cases. Various forms of trauma may cause a stenosis difficult to decanulate or extubate. Trauma, during the procedure of intubation, is very often charged with producing stenosis, which prevents the abandonment of the intubation tube. In my experience, this is exceedingly rare, the stenosis being due to other causes incidental to the disease for which the intubation is done. Diphtheria is essentially a necrotic process, that with or without intubation is apt to leave cicatrical stenosis, and, in my experience, stenosis has followed tracheotomy about as frequently as intubation. Decubitus is frequently referred to as though it were the presence of the tube that caused ulceration. I do not believe that a properly fitting intubation tube will cause any ulceration, no matter how long it remains if it be free from roughness or sharp edges, and is removed sufficiently frequently to be cleaned. Of course it is possible that trauma may be caused by insertion, and that this may be followed by local infection and ulceration, especially in the presence of infectious conditions, but I believe such trauma to be very rarely a cause of the stenotic cases we see. Tracheotomy is so commonly postponed until the very last moment that it is most frequently an emergency operation. Consequently the incision in the trachea is often very much misplaced, running off at an angle, or even slicing the side off the trachea like a slab from a log. Often also damage is done with dilating forceps tearing through the interannular tissue, and at times even denuding the cartilage of the rings. Then again, for one reason or another, various newly devised incisions, giving trap doors and even

excisions of cartilage are tried experimentally, nearly always resulting in more or less stenosis after cicatricial contractions set in. (Fig. 10, Case 10.) Undoubtedly in the insertion of a tracheotomy canula, especially if it be done without a pilot, it is very easy to denude the posterior wall of the trachea, and in time an ulceration may follow, which may be attributed to decubitus, when really it is simply oft-repeated trauma. A properly fitted tracheotomic canula should not produce decubitus, or even erosion of the epithelium. In this connection it may be well to state that nearly all the tracheotomic cases that come in to my clinic have been neglected, because it does not seem to be realized by most men that absolute cleanliness is essential, and that the tracheotomic outer canula ought to be taken from the wound and the wound cleaned at least once a day with additional changes of dressings as frequently as necessary between times. Neglect of this produces diseased granulations that result in building up a great mass of inflammatory infiltrate, which later becomes fibrous, and a thick dense scar results. Operations for malignancy and other conditions in the neighborhood of the trachea and larynx may cause stenosis, and in one instance I have seen a compression stenosis in the trachea due to cicatricial contraction following a burn with a band of hot iron externally on the neck. Attempted suicide occasionally results in serious damage to the cartilage, and if very careful work is not done, stenosis may follow.

Treatment of Organic Stenosis. The treatment of all the different forms of stenosis in much the same if cicatrices have formed. In cases which have not yet cicatrized, cicatrization must be brought about by excision of exuberant granulations and argyrol applications, as the very first step. The fungating granulations from necrotic cartilage are particularly troublesome. In these cases the quickest and best method is to lay open the larynx and trachea to facilitate drainage and resorcin applications. Such cases require laryngostomy anyway, but dilatation must not be commenced until the cartilaginous necrosis has ceased and healing is complete. In in-

tubated cases which show a tendency to close within a few hours or a few days after the removal of the intubation tube, it is safer, I believe, to do a tracheotomy and remove the intubation tube. In a few instances, however, it may be well to try the use of excessively large intubation tubes and gradually increasing sizes, great care being taken not to cause traumatism, and this treatment, I believe, had better not be attempted in the presence of ulceration. It is best adapted to comparatively recent cases of sub-glottic swelling. This form of prolonged intubation, with over-sized tubes has yielded good results in a few cases of cicatricial stenosis. Theoretically, it should yield better results than it does clinically, because the longer cicatricial tissue is held on the stretch the less tendency it has to recur and conversely the shorter the duration of the stretching the more prompt the recurrence. Its percentage of cures, however, is sufficiently high to warrant giving it the trial. Von Schroeter's hard rubber dilators are better adapted to hypertrophic than to cicatricial cases, because they can be left in only for a limited time, and for these cases I prefer the divulser. To be of any benefit in cicatricial cases dilatation must be prolonged, and for this purpose nothing equals the large size intubation tube modeled after the tube of O'Dwyer. In cases where the coughing out of the tube involves risk of closure of the larynx with serious dyspnea, the sub-glottic retaining swell must be large, and in most cases author's personal preference is a device suggested by a patient who was referred to me by Frank Trester Smith for a luetic cicatricial stenosis. It was a great comfort to this patient to know that the tube could not be coughed out, and I have subsequently used the same plan in a number of other cases with similar good results. It is essentially the same principle as the screwed-in-stem Roger's tube, but it is made of silver-plated brass, which I think is preferable to the hard rubber. It was illustrated in the Laryngoscope, September, 1909. (Priority for the principle belongs to Schniegolow and Rogers.) In the intubational treatment of cicatricial stenoses it is quite essen-

tial that the tubes shall be of large size and that they shall be worn constantly. The size should be increased up to the point where it requires quite a degree of force for insertion. Patients will complain of some pain for two or three days after a sufficiently large tube has been inserted. For this insertion, as well as the extubation, I have found the apparatus devised by Max Thorner to be preferable to that of O'Dwyer. The lower end of the tube is slanted and no obterator is used. I am speaking only of the treatment of chronic laryngeal stenosis. For diphtheria and like conditions I have never seen any improvement on the original O'Dwyer apparatus. In cicatricial stenosis Delavan and Emil Mayer have had excellent results with prolonged intubation followed by endo-laryngeal removal of granulation tissue, and also with the use of Schroetter's hard rubber dilators. The author has tried the wearing of soft rubber intubation tubes, because of the effect of soft rubber in softening cicatricial tissue, but as the procedure is not safe without a tracheotomy, it is better to do a laryngostomy when the effect of soft rubber is desired.

Abscesses have been the cause of the stenosis in two cases sent to the author for decanulation. Necrosis of the cricoid cartilage during pneumonia, in one case, was the fundamental process. In the other direct study discovered an old abscess in the "party wall." Treatment of these conditions by the direct method is easy, once the lesion is located, but the location is not always easily determined unless careful search is made. Good adventitious bands can be formed in some cases with the triangular punch forceps. In deciding the method of treatment to be used in a given case, it is very essential that a very careful bronchoscopic and direct laryngoscopic examination be made in addition to ordinary indirect laryngoscopy. With the direct laryngoscope and the esophogeal speculum the party wall can be accurately studied. In many instances the presence of granulation tissue about the tracheal canula should be removed in order to determine to what extent this is a factor in the

stenosis. Occasionally a case is encountered where the nature of the stenosis which has required a tracheotomy has not been determined, and where it is exceedingly difficult to determine it. In some instances there is nothing to be seen but a large, smooth, rounded swelling on all sides of the larynx, suggesting tuberculosis, lues, or an inflammatory condition. In such instances, before planning a procedure, it was at one time necessary to do an exploratory thyrotomy, but since the development of the direct method where ample specimens can be accurately taken, I believe it is possible to make an accurate diagnosis of conditions, if not of their etiology, in every instance. A sliding punch forceps should be used for this purpose, the distal end being inserted between the cords and a large mass of the tissue removed, always avoiding injury to the cricoarytenoid joints.

Abandonment of the Canula. When a tracheotomized case reaches the stage, by whatever mode of treatment, when it is to be trained to breathe again through the mouth it is necessary to occlude the canula. The best method, in the experience of Dr. Patterson and myself, is to insert a rubber cork in the inner canula. It is quite unnecessary, with a properly fitting canula, to have a fenestrum in the tube. The fenestrum causes no end of irritation and favors the formation of granulation tissue. because it is impossible to always have the fenestrum in the lumen clear of contact with the walls of the trachea. It is, anyway, unnecessary, because plenty of air will pass the canula if it be of the proper size, and breathing can be unimpeded without a fenestrum. If an attempt is made to leave the canula out, no matter how well the patient may be able to breathe, there is apt to be panic, because the removal of the tube is associated with dyspnea in the earlier history of the case; and associated ideas, terror and nerve habit cause the panic. On the other hand, when a cork is used for two or three weeks the patient becomes accustomed to breathing through the mouth and realizes that he can do so. When this confidence is acquired there will be no panic. In most instances where laryngostomy has not been done the

fistula into the trachea will close very rapidly. This should be retarded all that is possible, and the wound should be packed firmly open until the tracheal cartilage has united completely. Otherwise, there will be a mass of granulation tissue projecting into the trachea at the site of the unhealed wound. In an acute case where a tracheotomy has been done for temporary stenosis, if the wound is allowed to heal promptly the cartilage will heal very, very slowly, and all the time will be throwing granulations into the trachea, as I had abundant opportunity to observe with the bronchoscope some years ago. These may become so large as to demand a second tracheotomy. Of course, the granulations can be removed bronchoscopically and resorcin or other applications thus made, but as a rule it is better in such a case to open the tracheal wound again and deal with it properly by getting healing of the cartilage first. In cases where the canula has been worn a great length of time the cartilage is, usually, covered with fibrous tissue and possibly some extension of the tracheal epithelium, but not enough to prevent a prompt union. It usually takes but a short period of packing until the tracheal wound is united, and the wound will begin to fill up from the bottom, when the packing will be gradually, of necessity, less and less deep, until none can be inserted. In laryngostomy cases, when the true object of laryngostomy has been accomplished in the epidermatization of the surfaces with cuticular epithelium, a fistula has been produced which requires a plastic operation for closure after the abandonment of the canula. Very often this plastic operation will be only partially successful, so that repeated operations are necessary, but in all instances the result can be accomplished. After the abandonment of the canula and healing of the neck, the patient must be kept under occasional observation for some months. A patient can hardly be considered cured unless at least six months have elapsed after closure of the neck, without any dyspnea supervening. True, recurrences are uncommon after the lapse of one month, but by six months we are sure that there will never be any recurrence of the stenosis. Unless the operator has a good organization of assistants and

nurses familiar with this kind of work, decanulation, like all laryngotracheal surgery, is apt to be very disappointing. The routine work of the hospital in this class of cases is attended with a high mortality, and the literature on this subject is thickly strewn with records of unrelieved asphyxia, and of patients found dead in bed, and of deaths on the street when unattended.

Laryngostomy. When all else fails in a case of cicatricial stenosis, recourse must be had to laryngostomy, and with proper patience in carrying out the treatment, I believe that it will cure every case, though the treatment may require months or even years. Formerly, in the stenosis due to chronically recurring papillomata in children, I also resorted to laryngostomy in intractable cases, but since perfecting the technic of direct removal, I find that persistence with the extirpation and alcohol applications, it is possible to cure every case, though it takes, in some instances, a year or more to accomplish a cure. In no case is it necessary to give a general anesthetic for laryngostomy. The skin should be infiltrated with normal salt solution containing a minute quantity of cocaine, a quarter of a grain to the ounce is sufficient, as the action on the nerve endings seems to be qualative and not quantitative, provided the injection is intradermatic and hypodermatic. Salt solution alone will not answer, but it matters not how little cocaine is used. For the anesthetization of the tracheal laryngeal mucosa, a 10 per cent solution of cocaine can be used in adults. In children, it is better to infiltrate the mucosa just as the skin was infiltrated, because of toxic risks. The only really painful step of the process is the clipping with the turbinotome, which splits open the trachea and larynx almost instantly. In the technic of laryngostomy, I have practically nothing to add to what I have previously published on the subject. The success of laryngostomy depends absolutely upon the care with which the after treatment is carried out. In no other department of surgery does so much depend upon the care and watchfulness not only as to the manner in which the dressings are done, but to anticipate and ward off deformity, diseased granulations,

ulceration and other factors that produce hyperplasia, for hyperplasia will produce scar tissue, which is certain to give trouble later. Furthermore, it is necessary to keep down the granulations, because it is only over the small, firm granulations that the epithelium can make headway. In some instances, laryngostomy will be successful so far as the larynx is concerned, while below that, at one or more points, there will be a stenosis which persistently contracts at each attempt to terminate the treatment. In such instances, it is usually better to allow the upper portion to close and to prolong the treatment only at the stenosed area with only enough of lumen above and below kept open to facilitate manipulation. These cases, as, indeed, all cases of laryngostomy, require prolonged studious care and anything like routine treatment will be certain to result in failure. In some cases, upon splitting up the larynx and trachea, fistulae discharging pus will be found. It is quite essential to get these drained and healed before attempting to use the rubber dilating drain, as the tendency of the rubber is to interfere with drainage of the fistulae and to increase the chondrial necrosis, which is probably the cause of the fistulae. The fistulae will usually heal with careful dressing and an improvement of the patient's general condition, which is often very much impaired from careless and infrequent dressings, leading to exuberant granulations and the absorption of the purulent toxins through the neighboring mucosal surfaces. For the reduction of exuberant granulations, nothing has yielded better results for me than resorcin. As a stimulus to epithelialization, the German preparation "scarlet-red" (Biebrich) in a sterile 20 per cent mixture with petrolatum, has yielded excellent results during laryngostomy, though I understand it has been somewhat disappointing elsewhere. But this, or any other preparation, is, of course, useless unless the granulations are kept small and firm and prevented from becoming exuberant by resorcin and very careful, frequent packing. One of the most common causes of excessive granulation is neglect of frequent changes of dressing. There has been no improvement in the method of dressing outlined by Dr. Patterson in a previous

article of mine. One case that I reported in 1909 as then under treatment, proved to be a perfect cure, so far as the maintenance of the lumen of the larynx was concerned, but the closure of the opening by plastic procedures required 5 operations, and the closure was finally affected by my associate, Dr. Ellen J. Patterson. After each operation, a small fistula would persist. Dr. Patterson discovered that it was the pressure during cough that forced a small opening and forced the secretion out, thus causing a leak. By doing a tracheotomy very low in the neck, the pressure on the plastic above at the site of the laryngostomy during coughing was completely prevented. After healing of the laryngostomy opening was complete, the canula below was removed and the lower wound packed in the usual way until it closed from the bottom up. This new tracheal wound, not being epidermatized, healed promptly in about 10 days. This patient, now two years after complete closure, and four years after the lumen was enlarged to the desired point, remains absolutely free of stenosis and has now a good, though rough, voice. (Fig. 1.)

Discussion.

Dr. Emil Mayer, of New York: I feel that all of us present owe to Dr. Jackson a great many thanks for the masterly presentation of his paper and of the cases he has so carefully watched; these are the cases which perhaps are the most difficult the laryngologist is called upon to treat. I look upon stenosis of the larynx as requiring more care than any class of cases I know of and I wish again to express my thanks to Dr. Jackson for his presentation—in fact, he never fails to present to us something new.

However, I wish to present a procedure which has been enormously successful in my hands and which I believe if practised by others will be attended with equally good results.

A boy, nine years old, was sent to the Mount Sinai Hospital, New York City, with an open wound in his trachea. He gave the history that two and a half years ago he had been operated on because of diphtheria. The parents were very anxious to have this wound closed. He was brought in early in June of last year and the surgeon then in charge split the larynx and

made skin grafts. These broke down and the tracheotomy tube had to be reinserted. When the case was handed over to me I found that I was dealing not only with cicatricial tissue but hyperplastic tissue as well. For some months Freeman's probes were used for the slow dilatation of the tracheal wound. At that time I received an instrument I had sent for, a suspension laryngoscope. This was placed in position and I was enabled to produce a rapid dilatation in an incredibly short space of time, dilating the larynx from the tracheal wound so that I could see just what I was doing. I subsequently placed in a small rubber tube, keeping the tracheal tube in position. This was left in one week and then a larger one was inserted and finally a still larger one. It was then thought that the lumen was sufficient to enable me to intubate the child. I used the newer O'Dwyer tube with Roger attachment which I described. I found that I could hold the tube in place without trouble by the screw piece attachment. The O'Dwyer tube was kept in position for six weeks and during that time the child breathed perfectly well and could swallow all kinds of liquids and foods. The child was then, at the end of six weeks, extubated. The patient was watched carefully day and night. On the second day, feeling that the child's condition was bad, I put in the tracheal tube. The day following I re-intubated the child, using the ordinary O'Dwyer tube. It is now ten (10) days since the tube had been in position, and the child is breathing perfectly well and I expect to get a perfect recovery in this exceedingly difficult case. I cannot speak too highly of the use of the suspension laryngoscope, an apparatus of exceeding value.

With regard to the treatment of scleroma, I have employed tracheotomy and then the X-rays were applied directly. One patient is alive to-day and with no evidences of recurrence. Recently I have been able to keep a patient in satisfactory condition by the employment of the bronchoscopic tubes.

Dr. E. Fletcher Ingals, of Chicago: In acute cases such as occasionally follow intubation for diphtheria I have had some experience, and in one case in following the use of the bronchoscope. In the cases following diphtheria I succeeded in introducing smaller tubes, which would usually be coughed out in a short time. In some cases where there is much swelling I think the repeated introduction of smaller tubes, decreasing the size as rapidly as practicable, is the best course to pursue.

Dr. Burt R. Shurly, of Detroit, Mich.: All present are to be congratulated upon being here to listen to the masterpiece on a subject that so interests us. These are the cases that call

for our most strenuous efforts, perhaps, and especially a large amount of nerve strain and many emergency calls, where no well qualified assistant is at hand to do intubation work. In my observation the younger men are less skilled as the days go by, because fewer cases are met with now in practice. During the past year I have had two exceedingly trying experiences with cases of this sort, and both being apparently the result of traumatism. These two cases were distinctly traumatic in so far as the work performed by those absolutely unskilled in intubation. In one of these cases, a 3-year old child was intubated with a 5-7-year tube, the tube was introduced into the larynx with terrific force and was left in fourteen (14) days before the case was brought to my observation. On extubating the tube, the larynx I found was so closed, so stenosed, that it seemed advisable to do a tracheotomy, which I did. The child is now going through the procedure of decannulation. Of my acute cases following intubation, seventeen (17) in number, my experience agrees with that of Dr. Jackson's. Sixteen (16) recovered after a period of three or four weeks with rapid intubation with smaller tubes. These tubes were covered with 20 per cent. argyrol, or saturated alum in vaseline.

Dr. Harmon Smith, of New York City: I have had eight cases of multiple papilloma of the larynx in children, some of which have been treated by fulguration, but in nearly all cases the tracheal tube has been likewise used, so that correct inference relative to fulguration cannot be drawn. In one case particularly, where the tube had been worn several months, one application of the fulgurating spark resulted in the complete disappearance of the growths, which result led me to believe that fulguration would be a quick and sure method for the eradication of these growths. Subsequent attempts, however, have proved that whereas the growths will disappear temporarily from the application of the spark, they recur with almost the same persistency as when removed by operative measures, although not so quickly nor in such great quantities. In my last series of cases, consisting of four, misfortune has attended three of them. One developed diphtheria and died, another extracted the tube from the trachea during the night and suffocated before the house surgeon could reach him, and the third developed measles and was removed from my observation, although the larynx was almost entirely free from growths at the time of removal. In the fourth case, although having worn a tracheal tube for a year and a half, the growths have entirely disappeared from the larynx under the application of the fulgurating spark, and nothing but a fibrosis re-

mains. In one case of multiple papilloma of the larynx in a child, of specific origin, the growth disappeared under one injection of salvarsan, leaving the larynx completely clear, although considerable fibrous thickening remained, which prevented a clear and resonant voice. With regard to the subglottic thickening Dr. Lynah, of New York, has advised the use of a tracheal tube with a large fenestrum in the back for the purpose of admitting a certain amount of air at the side of the tube. By carefully grading these and with frequent changes the fibrosis had disappeared. Dr. Lynah also claims that in apparent paralysis of the larynx following diphtheria it is not a true paralysis, as he has examined many of the recurrent laryngeal nerves and found them to be perfect throughout their course, but that the lack of function is due to fibrous thickenings. With regard to rhinoscleroma, in addition to the use of the X-ray, Dr. Guntzer, of New York, has employed vaccines made from the dead bacilli of Frisch and Friedlander, and in those cases where this has been employed there seems to be no progress in the course of the disease. In the five or six cases coming under my observation in the past several years at the clinic of the Manhattan Eye, Ear and Throat Hospital those cases treated with these bacilli, together with the application of the X-ray to any external manifestation of the growth, there has been little or no advancement of the laryngeal scleroma and an apparent cure of the external involvement. These injections are made at various intervals, varying from two weeks to two months, depending somewhat upon the reaction that follows.

Dr. Emil Mayer, of New York City: Were some of these cases of fulguration done under anaesthesia?

Dr. Harmon Smith, of New York City: The majority were performed under chloroform anaesthesia as a primary measure, but the spark was not applied until the lungs had been thoroughly freed of the fumes.

Dr. Emil Mayer, of New York City: In these cases I am afraid of the spark igniting the fumes of ether.

Dr. Harmon Smith, of New York City: I always wait until the fumes of the chloroform have been expelled entirely from the lungs and I feel there is no danger from the spark; in the experience of a colleague of mine where ether was employed and sufficient time had not elapsed for the expulsion of the ether fumes from the lungs, an ignition occurred but no serious consequences resulted.

Dr. Chevalier Jackson: One of the first to observe and treat post-typhoid laryngeal stenosis in this country was Dr. Mayer, and I am glad to hear of his continued success with intubational

treatment. His last case was a difficult one because of the vicious cicatrices following his predecessor's attempts, and he is to be congratulated on his result. Dr. Ingal's remarks on the narrow neck intubation tubes appeal strongly to me, and the results of his ripe experience are interesting. Detroit has for years been a hot-bed of diphtheria, and few laryngologists in this country have done as many intubations as Dr. Shurly. I quite agree with him in the several points he makes.

PRIMARY CARCINOMA OF THE EPIGLOTTIS WITH REPORT OF A CASE.

BY EMIL MAYER, M.D.

Primary carcinoma of the epiglottis is a rare disease; Semon[1] in his review of cancer of the larynx states that he found primary disease of the epiglottis five times in 56 cases, while Fauvel, whom he quoted, found it but once in 37 cases of laryngeal disease.

Jurasz[2] mentions three cases of cancer of the epiglottis: 1, Man, 62, dysphagia, dyspnoea for one and one-half years, ulcerated tumor on epiglottis, size of a walnut involving the right ary-epiglottic fold. 2, Man, 50, dysphagia, dyspnoea, hoarseness, large cauliflower growth on site of epiglottis involving entire entrance to larynx. 3, Man, 45, sarcoma at base of epiglottis. On the right side it involved the false cord.

Schotz[3] records a case of cancer of the epiglottis the size of a walnut, confirmed by pathological examination; after surgical intervention (method not stated), there was a recurrence in three months and rapid increase in size with involvement of the lymph nodes along the carotid.

H. T. Butlin[4] presented a case of probably malignant disease of the epiglottis and the right half of the larynx.

H. K. Pel[5] mentions a case of carcinoma of the epiglottis in a man aged 51.

Adolph Bronner[6] showed a case of epithelioma of the epiglottis to the London Laryngological Society; this was a male 78 years old. The growth had been partially removed by biting forceps. The writer called attention to the rarity of these metastases.

Rosenbaum[7] treats the subject from the standpoint of external operation.

Angelesco[8] reports a case of epithelioma of the epiglottis

in a woman aged 68. Subhyoid pharyngotomy and death on the twelfth day.

A. Baurowicz[9] records a case of carcinoma of the epiglottis.

Harner[10] reports a case involving the epiglottis and right ary-epiglottic fold.

Flatau[11] records a case of carcinoma of the epiglottis in which lues was also present.

Spencer[12] presented a case of carcinoma of the epiglottis involving the base of the tongue.

Arnold Schiller[13] reports two cases of primary malignant tumors of the epiglottis. First, tumor the size of a walnut, involving the entire epiglottis, a mixed tumor, sarcoma and epithelioma. External operation. Recurrence six months later. The second case, epithelioma, death from pneumonia.

Chartres Symonds[14] presented a case of epithelioma involving epiglottis, base of tongue and glands of neck.

Brindel[15] reports a case of sarcoma of the epiglottis in a male aged 69. He says, "Although tuberculosis in all its forms, including lupus, occurs with predilection in the epiglottis, it is rarely the site of neoplasms. Malignant tumors exclusively limited to the epiglottis are rare, epithelioma being most frequent. It has the appearance of lupus, and attention is called to the likelihood of mistaking it for tuberculosis. Sarcoma is rarer than epithelioma, and may attain large size before attention is drawn to it. The symptoms accompanying epithelioma are apt to be dysphagia, pressure symptoms, salivation, fetid breath and modifications of timbre of the voice."

In Brindel's case an enormous tumor was discovered at the entrance to the larynx obscuring the view. The neoplasm was removed with the cautery loop. There was a recurrence in the velum palati, which was then removed.

A. C. H. Moll[16] demonstrated a case of squamous celled epithelioma of the epiglottis in an old man; external operation and removal of epiglottis with cautery, a cure is claimed.

C. Chauveau[17] reports a case of sarcoma of the epiglottis. This occurred in a male, aged 48 years, who consulted him because of a gland on the right side of his neck. There were no complaints except a slight disturbance in deglutition; a

small tumor was found on the edge of the right side of the epiglottis covering its laryngeal face. Histological examination revealed sarcoma. Patient disappeared when operation was proposed.

Delavan[18] records a case of primary epithelioma of the epiglottis. One-third of the epiglottis was removed by Krause with the biting forceps.

Pursegle[19] says that careless carcinoma is the most frequent form, then sarcoma, and lympho-sarcoma least frequent.

Lambert Laek[20] presented a case of epithelioma of the epiglottis and base of tongue, as also did Desante.

Jaboulay[21] records a similar case.

Horne[22] presented the case of a man, aged 43 years, who had dysphagia for three months, the right half of the epiglottis was injected and it was suggested that it might be malignant disease.

Lenhof[23] reports a case of cancer of the epiglottis in a male, aged 61 years, the patient complained of discomfort in swallowing. On the upper and right wall of the epiglottis and extending over the laryngeal surface is a reddish-gray flat raised area. The patient disappeared, returning three years later with a cancer on the right edge of the epiglottis extending to the base of the tongue. External operation.

Patterson[24] presented a man, aged 40 years, who had bad pain for two months on swallowing solids, this pain extended from the region of the tip of the great cornu of the hyoid bone to the ear. For a month past there was pain in swallowing fluids, the growth involved the anterior and posterior surfaces of the epiglottis, and was spreading to the tongue and the aryteno-epiglottic folds. There was cervical glandular involvement on both sides, which latter was believed to be due to bad teeth.

E. J. Moure[25] records a case of epithelioma of the epiglottis in a male 71 years of age. External operation.

A review of the cases cited shows how very rare primary carcinoma of the epiglottis is, especially when limited to that portion of the larynx.

Up to the present time the treatment has consisted of sur-

gical intervention or the use of radium and the X-ray. Of the former Vacher[26] states that "ablation of the epiglottis has been practised by removal with punch, scissors and cold wire snare. These methods are inconvenient. Hemorrhage may occur in spite of cocaine and adrenalin; it requires time and often repeated operations.

The heated loop answers every need, every precaution is to be taken that there be no short circuit and the epiglottis be crushed rather than extirpated, this latter causing danger of suffocation or severe hemorrhage. A curved guide covered with a heavy varnish is needful to hold the loop in position. The loop is of the thickness used in ablation of tonsils. The interrupter should be in the handle for control. If the epiglottis is too large for the loop, an incision is made in the center with a galvano-cautery knife. He does not find reaction greater than if removed with a punch, and calls it the operation of choice.

R. Helot and J. Payenneville[27] treated a case of epithelioma of the epiglottis by X-ray with the tube in the patient's mouth, under cocaine anesthesia; treatment lasted eight months. The tumor diminished in size and then remained stationary.

An interesting question arises as to the act of deglutition after removal of the epiglottis. The functions of the epiglottis and of the glosso-epiglottic fossa have been carefully presented by T. P. Anderson Stuart.[28]

"In the third stage of deglutition the arytenoids are opposed, rotated so that their vocal processes come together and incline forward. They glide bodily forward, so that their tips slide down the epiglottis and close the aditus laryngis.

"The larynx is drawn upward and forward, the epiglottis is pressed between the larynx and the base of the tongue, the glosso-epiglottidean fossae are emptied and the pyriform sinuses obliterated. The bolus glides from the tongue to the laryngeal surface of the epiglottis to the back of the arytenoids and into the esophagus."

Experimenting on animals, he found that when the epiglottis was held up by a thread there was no change in the movement of the larynx. The animals swallowed perfectly

well and there was little or no movement of the epiglottis. What movement there was seemed to be dependent upon movement of the tongue.

He mentions two cases to show that the arytenoids close the larynx and open the esophagus and that the epiglottis has no place in the swallowing act. Iredell mentions a case in which a pin lying from before backwards in the larynx of a girl of twelve years transfixed the base of the epiglottis with its point, while the head lay between the cartilages of Santorini. The head of the pin kept the cartilages from approximating, and thus swallowing was impossible. Quoting Poulet, he cites a case in which a needle lay with its eye in the left palato pharyngeal arch and its point in the left arytenoid cartilage. It prevented approximation of the arytenoids, and the patient could not swallow.

The writer has seen two cases of cancer of the epiglottis. In the first case the diagnosis was made as probable malignant disease of the epiglottis, and the subsequent history, although the patient passed from the writer's observation, was that of a primary epithelioma of the larynx, with subsequent metastasis, laryngectomy and death. The second case is here presented:

Male; aged sixty-four years; consulted me on June 9, 1912; complained of some slight pain in swallowing for the past six weeks, especially after the swallowing of cold liquids. He described it as being more a discomfort than actual pain. He had had no previous illness and his general condition was good. He would not have paid so much attention to these slight symptoms had it not been for the fact that both his father and mother died at the same age as the patient was at present (sixty-four years). A sister and a brother had died of phthisis and two brothers of diabetes. An examination showed some edema of the uvula and numerous small white spots upon the tongue and inner surface of the cheek. Larynx normal. His condition was diagnosed as leukoplakia buccalis.

He gave no antecedent history of lues. His condition improved under treatment, and in the following month he went abroad. On January 19 of the present year I saw him again, when he reported that the improvement after previous treat-

ment had not lasted very long and that he had the same discomfort after swallowing cold liquids. I found then that the uvula was normal in appearance; there was much nasopharyngeal catarrh; the white patches were still present, but no other evidence of any diseased condition in the throat. About three weeks later, on the 8th of February, he consulted me again, and this time I found a new condition on the laryngeal surface of the epiglottis. In its centre there was a deep ulceration surrounded by a thickened mass. I promptly removed a portion of this, and received the following pathological report: "Cellular tumor of an epithelial type, showing here and there a slight alveolar arrangement. The superficial squamous epithelium of the mucous membrane does not seem to be involved in the production of the tumor, as the individual cells are of the cylindrical type. Mitotic figures are very numerous. Diagnosis: Cylindrical cell carcinoma. (Signed) Dr. F. S. Mandlebaum."

We have then before us a case of carcinoma of the epiglottis in its very earliest stages of presentation, as the breaking down and induration had all appeared within a fortnight's time.

In order to be quite sure that this was the original site of the cancerous disease the patient was admitted to Mt. Sinai Hospital and subjected to a very thorough examination. This procedure occupied a week's time, and no diseased condition was found in any individual organ of the body. As a matter of routine blood tests were made, and here we received the first notification that the Wassermann test was strongly positive, although the patient gave no history whatever of lues.

On February 20 the patient was anesthetized and the suspension laryngoscope was placed in position, without, however, including the epiglottis. The epiglottis was now seized and with a long-handled knife was amputated, cutting from right to left and then from left to right. In this procedure I had the able assistance of my associate, Dr. Sidney Yankauer.

There was very little bleeding at the time of operation, and the patient was returned to his bed in reasonably good condition. On the following day (twenty-six hours after operation) he began to expectorate large quantities of blood,

and this was fortunately checked by sprays of peroxide of hydrogen, ice internally and externally.

Within a very short time the patient was able to swallow, soft foods at once, liquids later, and in ten days from the time of operation he left the hospital. Before leaving the hospital, however, he received one injection of salvarsan. One week after his return to his home he was able to attend to his affairs, and has been entirely well up to the present time.

From the foregoing it will be noted that the writer was enabled to see the very beginning and note the development of a carcinoma of the epiglottis. An early operation was permitted and the organ removed entire through normal tissue. It is the first operation of its kind performed under the ingenious suspension laryngoscope of Killian, and it is earnestly hoped that there will be neither recurrence nor metastases.

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Discussion.

Dr. J. Gordon Wilson, of Chicago, Ill.: There are one or two interesting points I would like to speak about. Considering how abundant the lymphatics are in the hypopharynx I consider that Dr. Mayer is to be congratulated in getting the case sufficiently early to prevent metastases.

I am especially interested in Dr. Mayer's remark in regard to the function of the epiglottis in swallowing. As Dr. Mayer said the work of Anderson showed how negligible was the action of the epiglottis in swallowing. Was it a useless organ? There is abundant proof that removal of the epiglottis does not affect the power to swallow. What then is its function? Some years ago I published a paper which has some bearing on this point. I then (Wilson Brain, 1905, p. 339) showed how abundant, and anatomically perfect are the taste buds which cover the posterior part of the epiglottis. In that paper I pointed out that here the primary function of taste, namely for protection, is preserved so the smallest trace of food or liquid passing over its surface immediately results in a reflex closing of the respiratory tract. This is one of the functions of the epiglottis. Another function less important in the adult than in the child, more important in the lower mammals than in man, is that respiration and drinking may go on at the same time. In the lower mammals the epiglottis passes up behind the soft palate so that it is possible for liquid to pass along the side of the epiglottis into the oesophagus while respiration is unimpeded.

Dr. Lorenzo B. Lockard, of Denver, Colorado: I am very much interested in the operation described by Dr. Mayer. I have operated upon a large number of patients, using the electric snare, the cold snare, and the Jurasz and Spiess forceps, but have never been able to remove the epiglottis as completely as did Dr. Mayer in this case.

The specimen exhibited demonstrates most strikingly the advantage of suspension laryngoscopy over the endolaryngeal methods previously employed, and is a strong argument for its employment in removing laryngeal growths. In tuberculosis the complete removal of the epiglottis, however, is not necessary, for resolution will occur even when only the upper

portion of the organ is amputated. In reply to Dr. Harmon Smith, I would say that I have removed the epiglottis in considerably over a hundred cases without ever noticing any difficulty in deglutition. The epiglottis, as far as is known, has no effect upon deglutition, and the only cases in which any trouble occurs are those in which there is also destruction of the portions of the larynx governing laryngeal closure: the arytenoids, aryepiglottic folds and ventricular bands. Even these cases are usually improved by the removal of the epiglottis. Sometimes, during the first few hours, there is some difficulty in swallowing, especially upon taking liquids, but this usually soon disappears. It is wise, however, for the first day, to have the patient drink through a tube, with the head bent forward between the knees.

Dr. Arthur W. Watson, of Philadelphia: I believe that the epiglottis acts as a gutter, as suggested by a writer whose name I cannot recall, to divert the mucus that drops from the palate onto the base of the tongue, and the saliva which flows over the base of the tongue, to the sides of the pharynx, thus guiding it around the entrance of the larynx. I think I have observed the result of the absence of this function in cases of extensive destruction of the epiglottis, manifested by sudden attacks of choking.

Dr. William L. Ballenger, of Chicago: Some time ago I removed the epiglottis for tuberculoma; there was difficulty in swallowing for two or three weeks, but this was not due to the removal of the epiglottis. The wound did not close by primary intention as expected. This epiglottis was removed deeply at the base. The difficulty in swallowing was more due to the external wound probably than to the removal of the epiglottis.

Dr. Emil Mayer, of New York City: closing the discussion: I would say the recurrence after removal of the epiglottis for cancer is usually slow. In one of my cases of cancer of the epiglottis there was no recurrence until after nine years. This case is an epithelioma of the epiglottis and the first on record removed with the suspension apparatus. The operation can be done more surgically and one need not cut into the malignant growth at all. The incision is made through the normal tissues. No part of the growth itself is injured. As to the removal of the epiglottis simply for a condition of paralysis this patient to-day is living in a perfectly comfortable condition. Whatever function the epiglottis might have, it does seem that our patients can get along well without it. I consider the suspension instrument of Killian as a great advance in our work.

NOTES CONCERNING THE SEAT OF LATENT DIPHTHERIA.

BY HENRY LEWIS WAGNER, M.D.

To the laryngologist engaged in the study of prophylaxis against infectious diseases an instance involving diphtheria of the upper respiratory tract will appeal with especial interest. It may be said in passing that the theory of the general ubiquity of the true Loeffler's bacillus has been thoroughly discredited. It is now indubitably established that diphtheria is spread only by an active germ-carrier coming into contact with healthy individuals under conditions which, though as yet undetermined, we have agreed to group under the somewhat vague designation "predisposition."

Diphtheria carriers may in general be divided into two classes: (a) Those who, though they have never been afflicted with the disease, yet accidentally carry the germ within their upper respiratory tract. These individuals, for the most part nurses and relatives surrounding the patient, we may call secondary, or "accidental germ-carriers" (Conradi's Nebentraeger). This class does not, as was formerly supposed, spread the disease.

(b) Primary or "active germ-carriers" (Conradi's Hauptraeger), i. e., convalescents and persons who, though now apparently healthy, have at some time or other been afflicted with the disease. These individuals, though months or even years may have elapsed since their recovery, still harbor the active bacilli within their upper respiratory tract.

Various have been the methods employed to exterminate the harbored bacilli of the active germ-carriers, but success has been difficult of attainment and in many cases the results have been absolutely negative. Briefly to pass in review some of the methods resorted to: In some instances carriers have been re-immunized with either toxines or antitoxines.

More recently a combination of both toxines and antitoxines has been employed. Again, the immunizing serum has been applied directly to the nose or throat. In still other cases various chemical agents have been employed as disinfectants. In Lyons, Rendu has resorted to the application of dry hot air—probably with the intention of producing a localized leucocytosis. Mention, too, must here be made of the manner in which the cultures of Pyocyanase or Staphylococcus have been utilized. Very recently tonsillectomy has been performed and, in cases where the faucial tonsils harbored the Loeffler bacillus, this method has yielded the quickest and most satisfactory results.

As will be observed, the greatest importance attaches to the exact ascertainment of the seat—the location of the germ focus. This locus foci may exist in the nose, in the tonsils, in a decayed tooth, in the salivary glands, or in any other well defined place within the upper respiratory tract. The present writer, with the assistance of associates, undertook a somewhat systematic search among a small number of carriers, but with results none too gratifying.

In a majority of the active germ-carriers under observation no special locus foci could be located. In only three cases was the germ focus found to be in the faucial tonsils. In one instance the nose was found to be the seat of the disturbance. This last case showed a well-defined caries of the middle turbinal. As I have been unable to find a similar case recorded in literature perhaps a brief sketch of it may be of interest to rhinologists.

The patient, a young man at school, contracted a primary nasal diphtheria involving especially the left side. Eight months later he developed ordinary tonsilitis, affecting particularly the left side. It was a Loeffler's bacillus and staphylococcus infection in both instances. In the interval between the diphtheritic attack and the tonsilitis, active diphtheria bacilli were found only once, and then only at the beginning of the interval—the patient being under observation during the entire time. Both times the patient was given large injections of antitoxin. The second series of injections,

however, was divided and graduated in what proved to be a successful effort to avoid anaphylaxis.

Recovery was complete, except that the left nasal passage now showed caries on the median line of the middle turbinal, and a resulting fistula 5 mm. long leading into a small ethmoidal cell. Notwithstanding the presence of the decayed bone, no odor of decomposition could be detected. There was no granulation tissue near the entrance of the fistula or within the ethmoidal cell. Active Loeffler baccilli were again encountered and persisted until radical interference annihilated this locus foci.

The total absence of any pathological sign (such as paresis, or paralysis of the muscles, formation of membranes, profuse secretions, either serous or purulent) is characteristic of nearly all active diphtheria carriers. A certain class of active carriers, however, such as improperly nourished children, show a sero-purulent—at times, sanguinous discharge from the nasal cavities. This purulent condition, however, cannot be attributed to the action of the Loeffler baccillus, but rather to the germs making up the original mixed infection (staphylococci and streptococci).

Discussion.

Dr. George L. Richards, of Fall River: The very curious thing about latent diphtheria occurring in the upper air passage is that the patients themselves are not really sick, but they give the disease to others in a very virulent form. I had a child in my office recently with a membrane on one side and nowhere else. The pathologist reported a pure culture. This child might not have been taken to a doctor at all. Yet this child was a danger to the community.

Dr. John N. Mackenzie, of Baltimore: Dr. Wagner has brought out a very important point. This pertains to the squeezing out of the pus, and the results of the bacteriological examination.

Dr. John O. Roe, of Rochester: The subject of Dr. Wagner's paper is a very important one. The results of his investigation of the nasal secretion very beautifully illustrate the importance of a bacteriological examination of the

nasal secretions, as well as those of the throat, in all cases of diphtheria, in order to be certain that the patient is free from contagion. Without this precaution, the patient might be a dangerous carrier of contagion when the examination of the faucial secretion might seem to indicate that the patient was entirely free from any such infection.

Dr. William E. Casselberry, of Chicago: I would like to ask if Dr. Wagner has been able to trace any particular line of communication by which the nasal secretions of the diphtheria carrier have gained entrance to the throats of others, thus spreading the infection.

Dr. Henry L. Wagner, of San Francisco, in closing: It is of great importance to urge the various boards of health to have in all cases of suspected diphtheria not only the secretions of the throat, but also those from the nasal passages bacteriologically examined.

CONGENITAL OCCLUSION OF THE POST-NASAL ORIFICES, WITH REPORT OF A CASE.

BY CHAS. W. RICHARDSON, M.D.

In July, 1912, there came under my observation a case of complete osseous obstruction of the post-nasal orifices at as early a period in the life history of the case as any other observer has seen such a condition, and at a much earlier period of life than have the majority of the reported cases come under competent medical observation. In an investigation of the literature on the subject I find only a few cases in which the obstruction was recognized in infancy.

According to Dr. R. Kayser, in Paul Hayman's Handbuch, the first case reported and operated upon in the living was made by Emmert in 1854. Otto, in 1830, reported the first case of closure of the choana in a foetus and next follows the interesting case reported by Luschka in a new-born dead child, 1859. Following these reports we find numerous cases of the condition reported from the anatomical table, the new-born infant and in child and adult life. It has not been possible for me to tabulate all the recorded cases in the literature, but I should judge that they do not exceed one hundred in number. It is interesting to observe that a fair proportion of cases has been reported by members of this Association. The obstruction of the post-nasal orifices may be membranous or osseous.

The membranous obstructions are usually formed posterior to the nasal cavities in the naso-pharyngeal cavity, but lay in contact with the post-nasal orifices so as to completely obstruct them. They extend from the upper surface of the soft palate at the union with the hard palate and extend to the roof of the post-nasal cavity.

The osseous obstructions are usually placed within the nasal

chamber within a millimeter or more from the free border of the posterior-nasal orifices. They are usually composed of thin plates of bone and may be unilateral or bilateral. To the two above mentioned forms may be added the congenital atresias by which the bones entering into the formation of the post-nasal orifice become united, thus more or less completely obstructing the post-nasal orifices. Various forms of atresias excited by disease and deformity, cause obstruction to the choanae, but, as these are not congenital, they do not enter into the scope of this paper.

On July 30, 1912, I was urgently requested to visit in consultation, early in the morning, an infant just born, who had marked difficulty in breathing. Dr. Wynkoop, who accompanied me on my visit to the infant's residence, told me of the nature of the case and the probable seat of the difficulty. When the infant was brought to me I was impressed with the peculiar cycle of respiratory difficulty with which it was contending. It would lie at rest for barely a moment, with its lips drawn tightly and its cheeks slightly indrawn, when it would begin to struggle for air, the face becoming suffused and slightly cyanosed, the condition being relieved as the child began to cry. As soon as its discomfort was relieved through the mouth breathing, necessarily instituted while it was crying, it would cease to cry, with a recurrence of the difficult breathing. The cycle was immediately suspended, when the lower lip was pressed down so that uninterrupted mouth breathing was permitted. Examination of the mouth and fauces demonstrated that all the structures in these parts were in normal relation and anatomically perfect. Examination of the nasal organ demonstrated that in external appearance it was normal. Inspection of the nares anteriorly, after removing the copious secretion, evidenced these to be in a normal condition—no adhesions or new growths. Washing out each cavity separately demonstrated that no fluid entered the pharynx, and after the cavity became filled with fluid it returned in excess by the anterior nares. The use of a probe proved that, at a depth of about five centimeters, the further progress of the probe was arrested by coming in contact with

a firm, rigid, resisting wall. The sensation imparted through the probe when introduced to the bottom of the nasal chamber was as though the end of the probe was impinging upon a firm, resisting plate of bone. The sound conveyed by the impact of the probe was similar to that imparted under similar conditions when a probe strikes upon a thin plate of bone thinly covered with soft tissues. It was impracticable, as well as considered unnecessary, to make an attempt to examine the post-nasal cavity through digital exploration, as the character of the respiratory difficulty and the examination through the anterior nares demonstrated, without doubt, that the deformity existing must be a complete obstruction of the post-nasal orifices. The maintenance of respiration until voluntary mouth breathing was established and the maintenance of nutrition presented problems to be considered in the management of this case. It seemed that an attempt at operative relief was only to be considered if the life of the child became endangered through its inability to maintain mouth breathing or through malnutrition. The operation itself, without proper protection in the naso-pharynx, might be attended with immediate danger, while the hemorrhage and resulting reaction were factors to be considered in an infant so young. Even should the operation have become imperative and the procedure have been successfully accomplished, the problem of preventing the closure of the artificially made opening in one so young would have indeed been very difficult. Through a relay of assistance, the child's lips were kept constantly apart so that continuous mouth breathing could be maintained, and, through this assistance, at the end of its second week of life, it had learned to voluntarily maintain mouth breathing, thus overcoming one of its great life problems. Its nutrition was maintained very satisfactorily by feeding it through the aid of a "dropper," thus solving the other problem. In a short period after establishing voluntary mouth breathing she learned to manipulate the bottle, alternating in sucking and breathing through the buccal cavity. From this time on the child has continued to develop as any child with normal respiratory organs.

The question to be considered in a congenital occlusion of

the choanae, especially of the osseous type, is the proper time for operative interference. The danger attendant and the difficulty of operating upon one so young, with the impossibility of maintaining the patulency of the opening after the obstruction has been removed, would seem to argue against an immediate operation if it be possible to maintain breathing and nutrition. The marked success obtained in the case that I present to you would seem to favor the expectant surgical policy in such cases, as well as the successful issue in many other cases. It would seem that the proper period for such cases would be as early as surgical interference could be done with perfect safety and with an absolute assurance that the manipulation could be made through a cavity large enough not to be embarrassing for want of size, and with a post-nasal space large enough to permit of easy introduction of the finger to note the obstruction from behind, as well as to afford protection and serve as a guide. The operation should nevertheless be done as early in life as possible, when the above mentioned conditions have materialized so that any alteration of the facial skeleton may be forestalled and before any pathological process has been started in any of the neighboring tissues or organs. It seems to me, therefore, that nothing having arisen to disturb the normal progress of a child with congenital occlusion of the choanae that about the second year of life would be the most appropriate time to undertake surgical interference.

Discussion.

Dr. William E. Casselberry, of Chicago: I have had three cases of congenital occlusion of the post-nasal orifices, one being an infant between three and six months of age. The first, an elderly man, had suffered through life from an accumulation of cheesy masses mistakenly diagnosed ozoena. The occlusion, total on one side and partial on the other, was caused by tense thin membranous webs across the choanae, which on being incised by a galvano-cautery knife electrode retracted, permanently freed the openings.

The second case, a man twenty-four years old, had complete unilateral occlusion by a bony diaphragm across the nostril about one-half inch anterior to the rear edge of the sep-

tum. Under general anesthesia an irregular aperture was made by three partly overlapping perforations with a Brainard drill. It was feared this opening might contract in healing, but examination some years later showed it freely open.

The infant was operated without anesthesia by a crucial incision through the membrane and soft bone which composed the diaphragm on each side. The openings remained patulous, the child thereafter nursed properly and grew apace, but succumbed to one of the infectious diseases a year or so later.

Dr. H. W. Loeb, of St. Louis, Mo.: I have had four (4) cases of congenital occlusion of the post-nasal orifices.

The first adult permitted me to drill on one side, but refused to allow me to operate on the other side, being content with what I had done.

The second case was one in which there was apparently a bony occlusion. He refused operation.

The third case was one in which the occlusion was not complete. There was a small round opening 0.5 cm. in diameter. This patient also refused operation.

The fourth case was that of a child suffering from asthma and who had a large mass of adenoids. He was operated upon with the result that he became free of asthma. About five (5) years later the child was sent back to me and an examination revealed bony occlusion of both nasal orifices. The child was then eleven (11) years old. The child died under chloroform. These subjects stand anesthesia badly.

Dr. Emil Mayer, of New York City: I believe that in the case of Dr. Richardson one can get a very good idea of the naso-pharyngeal condition by the use of the pharyngoscope; by its use one can ascertain the amount of post-nasal space, whether there is a narrow ledge of bone present or a complete obstruction. I recall one case in which I refused to do anything at all, not because of the presence of an occlusion, but because of the condition of the skull; the drill would have gone into the brain if extreme care had not been taken, and I was not willing to take such a chance.

Dr. Cornelius G. Coakley, of New York City: The anatomical arrangement which produces bony occlusions in these cases is frequently an outgrowth forward from the sphenoid, and the condition is a rare one. In one case I did a submucous resection, taking away the cartilage doing the typical operation for the removal of the pituitary body, and I got good results.

Dr. Charles W. Richardson, of Washington, D. C.: In closing the discussion, I would say that I reported the case because in the literature only about one-half dozen infantile cases have been reported, and most of them by members of this association.

I present to you a child nearly ten (10) months old who is very well nourished. I would like to show how well the child's nourishment has been maintained and without operation, and show a blackboard demonstration of the condition. In a child of this age it is, in my opinion, barbarous to make a digital exploration.

The main point is that in a child with both nasal passages stenosed it is possible to bring him to an age when an operation can be done with safety and with a certain assurance of success. In the child I present to you I think that possibly this coming summer, or the fall will be the proper time when he can be operated upon without danger to neighboring organs or tissues, and when the size of the naso-pharynx will readily permit of the finger being used as a guide.

The cases requiring operation in adult life are different. I will refer to a case of a girl sixteen years of age with an absolute unilateral stenosis. There is no question about my having opened up these cavities and there have been no recurrences. Also in a case of bilateral stenosis on a young adult there is no difficulty in the operation.

THE TRAINING OF THE SPECIALIST.

BY THOMAS J. HARRIS, M.D.

The last half of the 19th century witnessed a marvelous transformation in all walks of business. New methods and new means inspired by such master minds as that of Morgan were in evidence on every side. This development and expansion were not limited to business alone. They were equally apparent in every department of science. It was said of Goethe that his was the last great mind able to grasp all knowledge and all literature. In this wonderful advance medicine has borne a conspicuous part. There was a time when a physician could hope to master the entire realm of medicine. This has all changed, and this change has largely taken place within the last fifty years. Most, if not all of this progress, embracing discoveries which have revolutionized the conception and treatment of disease, is due to those who in the truest and broadest sense were specialists. Specialism in medicine and specialists have come to stay, like it or not as we may.

Among the earliest, if not the earliest, to attain to the dignity of being a specialty, was that of laryngology. Horace Greene, denounced and condemned by his contemporaries, may be properly considered one of the specialists in laryngology in this country. His active work was done only a little over fifty years ago. This association was founded in the year 1878, and embraced on the roll of its founders almost the entire number of those who were devoting themselves at that time to the specialty of laryngology in America. Consider the marvelous expansion since that day, both in the number of specialties and in the number of men, properly or improperly, announcing themselves as specialists in laryngology. The 17th international Congress of Medicine, to be held in London this coming August, announces 23 sections or departments in medicine. In place of the little group of twenty or thirty men in

1878, if we are to take the estimates given by competent authorities, there are in this country to-day alone, several thousand men described as laryngologists. In the State of New York a list has recently been compiled, numbering several hundred.

A perusal of the early volumes of the transactions of this Association will show the magnificent work that was done by the laryngologists of thirty-five years ago. The specialty, however, has extraordinarily developed, and to-day it means vastly more than it did at the time of the establishment of this Association. No longer is it confined to the treatment of diseases of the larynx. The closely associated and all-important field of rhinology has been discovered and demands equal or even more careful consideration; and, while not actually a part, in practice otology, too, has become so wedded to laryngology that it is usually considered to belong to "greater laryngology." Bacteriology, with all that it means, has been born since the early days of laryngology, as well as the important field of bronchoscopy.

This rapid expansion and the increasing numbers of those who are announcing themselves as specialists raise a most serious question, and one which is deserving the careful consideration of this Association. It is, What preparation or education is required in order to become properly equipped for the specialty? Already it has been the subject of an interesting discussion before the American Laryngological, Rhinological and Otological Society, and last year it was considered at the Liverpool meeting of the British Medical Association. As Shambaugh rightly points out, a proper training should give a man the ability to do not merely routine, but original work, something that we in this country have been sadly lacking in.

It cannot be emphasized too strongly that the clinical side is only one side of a specialty. It has been my privilege for many years to be engaged in postgraduate instruction, and I have had in this way an opportunity of meeting many men who are proposing to specialize. More and more I am impressed with the inadequacy of the preparation that the great

majority make. The six-weeks specialist has been a constant reproach to postgraduate schools. This, however, is not altogether the fault of the school. The postgraduate school is only responding to the demand of the practitioner. If we are creditably informed, many a small town in the Middle West possesses no less than five or ten "nose and throat specialists." Where they acquired their specialty is an unknown quantity. As Wishart, in a communication later to be referred to, very forcibly states: "Up to the present, on this continent, there is no recognized portal to the pursuit of the specialty. On the other hand, the gates may be said to be many, and yet, *mirabile dictu,* our towns and villages, and even our cities, are filled with "specialists" who have entered by no gate whatever, but have simply 'climbed over the wall' and to some extent, at least, are to be considered merely as thieves and robbers."

The basis of all medicine is anatomy and pathology. This seems to be lost sight of by the man who too often appears to choose the specialty of laryngolgy because it is an easy way of practising medicine and making money. Only recently in Vienna, we were told of an American who went there to get an opportunity to do a series of sub-mucous resections, and who publicly asserted that that was all that he had come for, and that he had no wish or desire to acquaint himself with the anatomy or pathology of the subject. Only second in importance is a thorough training in general surgery. Our specialty has become to a large degree surgical, and we are called upon, as our predecessors never were, to perform all sorts of major operations. This demands a thorough grounding in every phase of surgery. Of general medicine and its profound bearing upon our specialty it is unnecessary to speak.

Only after the young man has been thoroughly grounded, then, in anatomy, pathology, surgery and medicine is he prepared to take up the clinical side of laryngology. This preparation cannot be made in a month or a year. To our mind, it demands at least two years and, better, three. We recognize, of course, the objection that will be raised in regard to the time required. This can be probably reduced to a considerable

degree by a more thorough preparation in the undergraduate school, although the kind of training which we have in mind can never be entirely acquired by the student who is studying for a degree and who must prepare himself upon a number of subjects. What are the usual methods of preparation for special work at present? They are, first, learning the specialty in the course of a general practice with or without connection with a special clinic. This is the method that many of the beginners in our specialty were compelled to pursue, and it continues to be the method pursued by many men who, in a natural way, graduate little by little out of general practice into some specialty. Usually the physician who is doing this is so occupied by the demands of his general practice that he can devote only a little time, and that with much irregularity, two or three times a week to the clinic, gaining some instruction from his chief, but compelled to learn for the most part by himself. The objections to this method are manifest. It can never give a broad training, although it is bound to produce what is of inestimable value—a man well fitted by years of experience for special study.

The second method is by taking postgraduate courses here or abroad. There has been a marked advance in the method of instruction in our postgraduate schools in recent years. A student can get excellent short courses in every department of laryngology, as well as opportunity to work in the various clinics as long as he may desire. To our mind, the value of the postgraduate medical schools cannot be overestimated. They have been and are doing a tremendous good for the physician. Their chief mission, in our judgment, however, is to the man in general practice who desires to brush up in order to do his work more efficiently. The four or six weeks' course should be entirely done away with, except for specialists who desire to do advanced work, and no beginner should be received for a period of less than six months, or, better still, a year, and then only after he has had the necessary groundwork in anatomy, pathology and surgery just referred to. This should take the form of connection with a laboratory and a surgical clinic, each for a period of at least a year.

Third, residence as interne or assistant in a special hospital. This offers, as far as the clinical side of the training is concerned, undoubtedly the best opportunities of acquiring the laryngeal opening. The effect of the frost bites, however, was special hospitals in this country or in Europe, residing there from one to two years, will undoubtedly come out well trained from the clinical standpoint. Such opportunities are, however, comparatively few, and even in those who have enjoyed them there is usually a sad lack of preparation in the groundwork to which we have just referred and which is so essential for a thorough training.

That this question of education in medical specialties is receiving the consideration of the profession is evidenced by the action taken at the recent Clinical Congress of Surgeons held in New York last fall, in regard to the establishment of a post-graduate surgical degree. If this is true of the surgeon, is it not equally true of the man working in other departments of medicine? Dr. Wendell Reber of Philadelphia, in a paper read before the Pennsylvania State Medical Association last year, on the teaching of ophthalmology in America, has advocated the giving of a degree in ophthalmology to men who have successfully carried out a required course of instruction. He believes that there should be some agreement between the fourteen postgraduate medical institutions in this country "as to the minimum training required of applicants for examination, and some approach to uniformity in the ordering of a three and a six months' course." We would, as we have said, favor a decidedly longer time than three months. "He recommends that no applicant for postgraduate teaching should be admitted to any of the well-recognized specialty courses who has not completed three years in general practice or its equivalent in general hospital work. If this idea should be carried out in connection with all the specialties in medicine, we would have in a few years a highly trained body of men to whom the public would turn as the men best qualified by education and experience to carry on their work." (Quoted from "The Medical Times.")

To these views in the main we heartily subscribe. What we

desire is *quality* in those entering our specialty, not *quantity*. The American method of getting results by the quickest means, together with the commercial spirit which too often obtains at the bottom, we must sadly confess, has not escaped the noble profession of medicine. Specialism, with all that it represents for good, has unfortunately tended to offer an attractive field for commercial exploitation. It cannot be disputed for an instant that there are too many specialists. The public, it is our honest conviction, would, if two-thirds of them should be removed by marrying a rich wife, or some other equally certain but pleasant cause, be the better for their removal. The inauguration of some form of examination requisite to a degree, would, beyond a doubt, work most beneficially in this direction.

We do not wish to give too pessimistic an opinion, but it is our experience, based on what we have seen here as well as abroad, that the man who has the proper idea of what specialism is, and what preparation for it is required, is a great exception. We can do no greater service to the specialty we love and represent than on every occasion to impress on those who have aspirations to specialize the seriousness of the undertaking. Individual action alone, however, will not be sufficient. A concerted advance movement to properly *standardize* our specialty is essential. In our opinion, the time is ripe for such a movement. As a result of the discussion before the American Laryngological, Rhinological and Otological Society two years ago, a committee was appointed to consider the matter. This committee has spent much time in obtaining the views of American and Canadian laryngologists, which they incorporated in a report which will be presented at the meeting of the society this week. This report, prepared by Dr. D. J. Gibb Wishart, chairman of the committee, I have had the privilege of examining. It agrees in the main with the views expressed in this paper, namely, that a definite plan or scheme of instruction for special work should be determined upon. As an outline for a more definite working basis, this should consist of the following divisions: First, a thorough preparation in the undergraduate medical school; second, a general preliminary training through six months of special instruction covering not merely the clini-

cal side, but the anatomical, pathological, surgical, etc.; fourth, eighteen months of interneship in a special hospital or in the ear, nose and throat department of a general hospital; fifth, at the conclusion, an exammination by a university which should confer upon the successful candidate a postgraduate degree, such as Ph. D. (Oto-Laryngology). The committee is strongly of the opinion that the postgraduate instruction should be given in some established university, and further, that to make the departure a complete success, legislation will be necessary. This, when pubilc sentiment has been aroused, there will be no difficulty in securing. Finally, the committee is of the opinion that any success in this advance movement must be the result of concerted action on the part of the several national societies representing America and Canada, and this Association will, in all probability, in due time receive a communication asking it to join in the further consideration of the proposed standardization.

Discussion.

Dr. Charles W. Richardson, of Washington: The Triological Society, of which I am a member, has many times brought up facts that bear on this same line, and I think that Dr. Harris' paper is a very timely one and should help to impress upon us that such a course as outlined is a very essential one. It is well known that in every community where it is possible to obtain superficial information along these lines in a quick way for emergency purposes, it is graspable. Such instruction is asked for by the under-graduate students. Often the under-graduate student comes to the teachers of special subjects and inquires about what specialty they should follow, and when these students are carefully questioned it is found they wish to take up this specialty, not because of any particular adaptability to do the work, but because such and such a practitioner has been able to follow it and make it a success. They look at it from a purely commercial aspect. The student is willing to prepare himself in the ground work in an under-graduate course, but he is not willing to take a course of instruction in a hospital, and at the same time acquaint himself with general medicine. He is not willing to devote one or two years in general practice in order to thoroughly equip him-

self in general medicine before taking up the study of a specialty; nor is he willing to give up one or two years, or even more, in the preparation of this special work. We all know that there are many men who attempt to specialize, but the work proposed by Dr. Harris is the work we should insist upon. Such a method as outlined by Dr. Harris is impressed upon us all the time. We overlook the fact that many universities require the presentation of such degrees as Ph. D. as being absolutely essential before the student is allowed to take up the study of such a course. This is a fact that should be drummed into the societies. As regards getting legislation on this point, I think Dr. Harris will find, as we in Washington found it extremely difficult in going before Congress and having passed ordinary, but necessary legislation required even for our own city's benefit. We had to fight to the utmost degree for medical legislation, fight for notification even to the last ditch. We had to fight even for a filtration plant and this was almost lost because of the so-called "lack of money." Universities can in my opinion, give considerable assistance by the proper alignment of the different schools in this line of work. Standardization is a very important point.

Dr. H. W. Loeb, of St. Louis: I think it is easy to find an explanation, especially in many of the smaller cities of the country. In this country there are one hundred and fifty thousand (150,000) physicians, and I hesitate to say how many of these are qualified to practice medicine in any branch. The requirements to-day are different from those of twenty-five (25) years ago. A few years ago the men with a grammar school education found it easy to undertake the study and practice of medicine. However, many of these men, after practising from five to fifteen years desire to share the supposed prosperity of others who specialized. One can not stop this class of men from becoming specialists in this country.

In the next twenty-five (25) years the properly prepared physicians will be in the majority. A man who has spent ten (10) years and $3,000 to $5,000 on his education will not waste his education by taking up a specialty after undue preparation. I am heartily in favor of any movement which tends to improve the situation, but this will be easy when the standard of physicians of this country is equal or superior to that of other countries and where the six weeks' specialist is practically unknown.

Dr. Burt R. Shurly, of Detroit: I will refer to one meeting of the A. M. A., which occurred in the Middle West, where it was stated that out of 6,100 practitioners, one-fifth had

registered as specialists in diseases of the eye, ear, nose and throat. This does not tend to show any high standardization of our specialty. In order to accomplish what is needed it seems to me that the formation of the American College of Surgery, formed last night, was a step in the right direction.

Dr. Max A. Goldstein, of St. Louis: I have been often impressed by the fact that the otological surgeon of experience will complete a difficult operation, having mastered its technique, etc., and yet he will allow the general surgeon to ligate, for instance, the jugular vein, an operation the ordinary country surgeon is qualified to do. The general surgeon might be called in to do a simple tracheotomy when the laryngologist, when called in to see the same patient in consultation, may see fit to do a laryngectomy, an operation of greater difficulty than the doing of a tracheotomy. A feature brought out in Dr. Harris's paper is that many men are not qualified to enter this field of work; they are deficient in advanced laboratory training. The best men to-day are those who are familiar with what the microscope reveals and who have proper and sufficient clinical training in a hospital that is well equipped for this. I think that all should express to Dr. Harris a voice of thanks for what he has presented, which is so timely.

THE REMOVAL FROM THE ESOPHAGUS OF A PLATE OF FALSE TEETH IMBEDDED FOR EIGHTEEN YEARS, BY MEANS OF AN ESOPHAGOSCOPE.

BY D. BRADEN KYLE, M.D.

With the aid of the esophagoscope it apparently would not be difficult to locate a foreign body in the esophagus, and in the majority of instances this would be true; but in the case which I wish to report, where the foreign body had been imbedded for eighteen years, the local conditions are so entirely different from those produced by a recent foreign body, that the entire procedure is different. The granulation tissue which had organized into fibrous tissue, the imbedding of the foreign body in this tissue, together with the curvature of the spine as shown in the X-ray, rendered it exceedingly difficult to locate the foreign body in spite of its size and shape. The age of the patient and his generally poor condition, were also important factors in this particular case, the history of which is as follows:

Early in the morning of October 15, 1895, the patient was suddenly awakened while dreaming—dreaming that he was swallowing egg shells. On becoming thoroughly conscious, he realized that he was actually swallowing some hard object, and investigation promptly disclosed the fact that an upper suction plate with four attached front teeth, which he had left in place on retiring, was missing. Inserting his finger into the throat, he was able to touch the plate, but in his effort to grasp it, only succeeded in pushing it further down.

A physician was called, made an examination, but could see nothing. As the patient could feel that the plate was lodged in the upper part of the esophagus, the physician wanted to attempt pushing it down into the stomach. To this procedure the patient objected, and then went to a hospital, where another physician examined him, passed bougies and probangs, but

with no result whatever, and ended by assuring the patient that he had never swallowed the plate, or, if he had, that it was no longer in the esophagus.

During the first three weeks following the disappearance of the plate, the patient experienced slight pain in the lower part of the neck, at the point where he always felt the object had lodged. After that time, there was practically never any painful sensation, but swallowing has always been difficult. When solid food—meat, potatoes, etc.—were taken, they had first to be finely ground. For months at a time, he took only liquids. At the time of the accident his age was fifty-five years and his weight 138 pounds. After about the second year, he began to notice he was losing weight, and continued to do so until the present time, when, at the age of seventy-two, he weighs but 110 pounds.

In 1901, six years after the disappearance of the teeth, the first X-ray plate was made in a further attempt to locate them. The plate appeared to show a slight shadow just above the cardiac end of the esophagus. The passing of bougies, however, failed to disclose the presence of a foreign body in that region, and again the search was abandoned.

The patient's general condition continued the same until about the middle of January, 1913, when the difficulty in swallowing beyond a certain point was markedly increased. On January 27, 1913, the patient was referred to me by his family, physician, Dr. W. H. Hartzell. An X-ray plate was at once made. The negative (lateral view of the neck) showed distinctly the plate, its long axis parallel to the walls of the esophagus, and located immediately behind, and extending a little below, the cricoid cartilage. The teeth were attached to the lower end of the plate.

The same evening the patient was taken to the operating room, and the removal of the teeth attempted. The pharynx and esophagus were anesthetized with 20 per cent. solution of cocaine, and the patient placed on a table with his head well extended over the end. The extension of the head on the shoulders was very difficult, owing to rigidity of the entire spinal column. An attempt was made to secure a view of the plate by

means of the Kirstein autoscope, but this was found to be too short, and the Kahler esophagoscope was used instead. With this instrument, a portion of the surface of the plate was brought to view, about eighteen centimeters below the anterior margin of the upper jaw, but the upper edge appeared to be covered with fibrous tissue.

Efforts were made through the esophagoscope with various forms of forceps, to find and grasp the edge, but without result. The esophagoscope was then removed, and an attempt was made with an ordinary long laryngeal forceps. I was able to partially displace the thickened tissue over the upper margin of the teeth, so that with the laryngeal forceps a hold was secured, but considerable force failed to dislodge the plate. It was then decided to allow the patient to rest for a time. Very little soreness and practically no hemorrhage followed this manipulation.

On January 30, a second attempt was made, the patient anesthetized as before, but this time sitting on a low stool with head and back supported by an assistant. This position was found to be much easier for both patient and operator. On this occasion, through the Kahler esophagoscope, a portion of the tissue covering the upper edge of the palate was cut away with biting forceps, thus exposing it to view. Seizing the plate with the same forceps, effort was made to withdraw it, but the forceps slipped off, and the plate remained. After similar repeated efforts, owing to the fatigue of the patient, it became necessary to again cease manipulation for a time.

In the meantime, another instrument, a long, biting forceps suitable for use through the Kahler esophagoscope, had been secured, and on February 4 a third attempt was made, in all respects similar to the second, except that the new biting forceps was used for cutting away more of the overhanging tissue and grasping the plate. The plate was distinctly loosened from its bed at this time, but not sufficiently to be withdrawn before weakness of the patient, and slight hemorrhage, which obstructed the view, made it seem advisable to discontinue a third time.

On February 10 the patient was prepared as before, and,

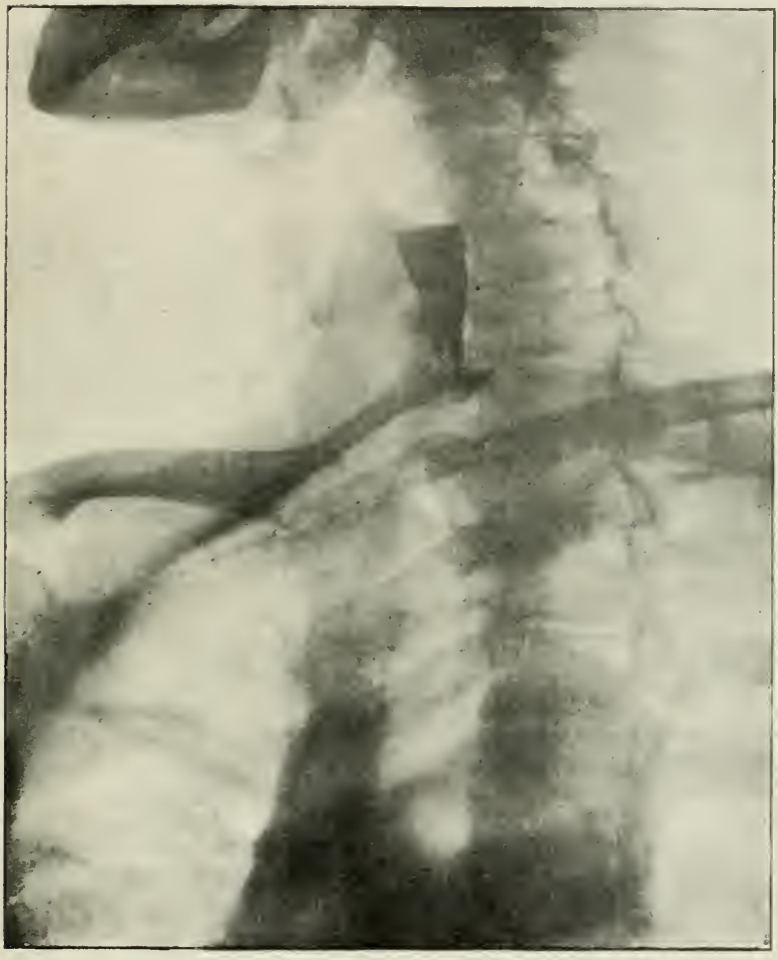
with the Kahler esophagoscope, the plate was promptly located and grasped with the biting forceps, which held while steady traction was made. The plate moved slightly—perhaps half or three-quarters of an inch—when the forceps slipped off. The esophagoscope was readjusted, the assistant supporting the head was directed to apply external pressure to the foreign body in a direction backward and upward, the plate was again seized, steady upward traction applied, and esophagoscope, forceps and teeth all steadily withdrawn.

The plate and teeth were found to be in a perfect state of preservation. The plate measured one and three-fourths inches in length by one and one-quarter inches in its greatest breadth. Practically no hemorrhage and very little soreness followed the final operation. The second day after the removal of the plate, difficulty was experienced in swallowing. This difficulty in swallowing was not due to any swelling, but to the fact that there was no muscular action in the esophagus and the portion involved by the foreign body was almost the same as the sacular dilatation; in other words, the cause of the lateral distention in the esophagus had not been removed and the physiological contraction did not take place. Liquids taken appeared to be arrested in the pocket which the plate had formed in the anterior wall of the esophagus. It was found, however, that by taking a very small portion of liquid at a time it could be swallowed without great difficulty.

Local anesthesia was used, first using a five per cent. solution of cocaine in the upper part of the esophagus, and then by means of an atomizer through the esophagoscope a twenty per cent. solution was used further down.

The scar tissue formation was not circular, but was limited to the point of impingement of the suction plate at its widest di ameter, as is shown in the X-ray. This made two areas of scar tissue, one on each lateral wall. However, at the top of the plate, on account of the friction, granulation tissue had formed and extended out over the upper margin of the plate. This tissue was almost a quarter of an inch in thickness and extended down over the plate a considerable distance. This scar tissue was removed at one margin of the plate, so as to enable me

to pass an instrument underneath the plate and in that way make a certain amount of traction and force the plate away from the scar tissue. As shown in the X-ray photograph, the



foreign body was on the laryngeal side, the curvature fitting around the larynx.

One of the peculiarities of the case was that the patient had not suffered from any laryngeal symptoms, and it would seem impossible to have a foreign body of such size imbedded in the

esophagus and against the trachea without producing any respiratory symptoms. In the early history of the foreign body, the patient complained of some soreness in his throat, although he never suffered any severe pain and never any difficulty in breathing. There was slight alteration in his voice, due more to interference with the muscles of phonation than to the inflammatory action. The esophageal muscles, having been put on tension for so many months and years, had lost their muscular elasticity, and the inability of the patient to swallow after the removal of the foreign body was due more to this than it was due to the amount of scar tissue.

It is now three months since the removal of the foreign body, and the esophageal muscles have increased considerably in their action, and the patient is now able to take semi-solid food.

I think the successful removal of this foreign body was due largely to the fact that I proceeded slowly and made as little trauma as possible in the esophageal structures, and by loosening the foreign body from its fibrous bed, by setting up slight inflammatory action and then waiting a few days, I was able to remove the plate without much laceration of the structures.

Discussion.

Dr. Henry L. Swain, of New Haven, Conn.: I wish to congratulate Dr. Kyle upon being able to recover the teeth after so long a period and also because the patient has not developed any malignant disease or other bad results because of its stay so long in the esophagus.

Dr. Algernon Coolidge, of Boston: I congratulate Dr. Kyle upon being able to remove the tooth plate without an anaesthetic. The principal difficulty in removing foreign bodies from the esophagus is often a deficiency in armamentarium. To cut tooth plates and similar work requires very special equipment. Large esophagoscopes are often of great service. It must always be remembered that the trachea may be compressed from behind by a shifting of position of a large foreign body, and it is well to have a bronchoscope at hand in case breathing becomes obstructed.

Dr. Kyle: In closing the discussion I would say that the curvature of the spine, as shown in the X-ray plate in this

particular case, rendered it very difficult to pass the esophagoscope. It was only by careful manipulation and continued patience that the operation was successful. The continuous looking through the tube is very tiresome to the eye, and I was fortunate in having a trained assistant on whose services I could rely, and I am indebted to Dr. Fielding O. Lewis and Dr. Arthur J. Wagers for such assistance.

FOREIGN BODIES IN THE ESOPHAGUS

Report of Two Cases.

BY CORNELIUS G. COAKLEY, M.D.

F. M., male, age 16 years, was admitted to Bellevue Hospital, Fourth Surgical Division, in the service of Dr. Thomas A. Smith, in the early afternoon of December 31, 1912. The patient gave a history that while eating soup for his dinner that day, a piece of the plate broke, and that he thought he ate a piece of the dish, and thought he could feel it in his throat. After being admitted to the hospital he was seen by the assistant visiting, who ordered an X-ray plate taken to determine whether there was any foreign body in the throat, as none could be seen or felt. On January 1, 1913, at 1:00 P. M., the assistant visiting notified me that there was a patient in the ward of the hospital, with a foreign body in the larynx, as seen by X-ray, had a temperature of 105 with a rapid pulse and suspicious pneumonic area in the right lung. I reached the hospital about 3:30 P. M. and found a full grown boy, sturdy build, delirious, scarcely able to talk. On examining the pharynx I found an edema extending from the vault of the nasopharynx as far down as one could see with a laryngeal mirror, or feel with the fingers. The amount of edema and swelling was such as to greatly resemble a retro-pharyngeal abscess. There was a very fetid odor to the breath, and an almost aphonic sort of crying. The X-ray plate showed a triangular shaped foreign body, the base of which was uppermost, horizontal, and at the level of the seventh cervical vertebra. It could not be felt with the fingers. Owing to the huskiness of the boy, and the very acute delirium, it was necessary to administer chloroform before passing the esophagoscope. With the esophagoscope I was able to detect, after a little difficulty, owing to the swelling in the upper portion of the esophagus, a piece of plate. It was grasped by a forceps

and withdrawn, followed by a small amount of bleeding. The boy came out from the anesthetic, continued delirious, coughing muco purulent fetid expectoration. The rales in his chest increased and patient died fourteen hours later from acute septic pneumonia. Foreign body triangular in shape, measuring 2.3 cm. long on one side of the triangle, 3.2 cm. long on the other and 2.2 cm. long on the other. It is very jagged and evidently had cut into the mucous membrane so as to rapidly infect the pharyngeal mucosa, and the secretion from the infected pharynx passing into the larynx set up a septic pneumonia within twenty-four hours. This case illustrates the necessity for prompt operative relief for the removal of sharp foreign bodies.

On going down to the ward the same day, I was asked to see case No. 2, F. S., male, age 3 years, who was admitted on December 31, 1912, to the Fourth Surgical Division, service of Dr. Thomas A. Smith. History was that three days before admittance, child had swallowed a coin, about the size of a quarter. An X-ray was said to have been taken at some other hospital, showing a coin at the level of the sixth cervical vertebra. Child had no symptoms, swallowed his milk perfectly, had no respiratory symptoms, and there was no change in voice. Attempts had been made to remove the coin, but whether it had been pushed down to the stomach or not, the staff did not know. I advised another radiograph, which showed the coin there, and on January 3, I removed the coin, a five centesimi piece, diameter 2½ cm. The child had no discomfort following the operation, left the hospital the next day and has not been seen since. Here was a smooth object in a small esophagus that did not even interfere with deglutition and had been in position five days before it was removed.

Discussion.

Dr. Emil Mayer, of New York: I here present a unique object which I have removed from the esophagus. It is a metal tag with the wire attached. The child was brought to my office in the middle of March with the story that he had had

a retropharyngeal abscess in December. After being cured of this he was better until he became hoarse and had some difficulty in swallowing. The child is one and a half years old. I got a good view of the larynx and saw a swollen epiglottis and thought it was due to some suppurative affair connected with the retropharyngeal abscess. The child was sent to the hospital and examined by Dr. Koplik, who thought he detected a sound of an unusual character and advised the use of the X-ray. The picture showed the presence of a foreign body which looked like a locket. The father was sent for and denied that the child had swallowed anything or that he had missed anything. The next day I removed without anaesthesia the foreign body which I present. The child died twelve hours after without any apparent reason. If the child had been seen earlier I believe that such a foreign body could have been removed without a fatal injury. An interesting point is that the father acknowledged afterward that on January 6 the child had swallowed something. The doctor who was called in said that there was nothing wrong. Another doctor who was called in made the diagnosis of adenoids. When the child was in a dying condition, he was brought to me. I should have been told the story about the foreign body.

Dr. Chevalier Jackson, of Pittsburgh: I myself and others have stated very frequently that it is a crime to pass any instruments blindly into the esophagus in an attempt to remove a foreign body. Unfortunately, a number of cases have occurred which have led us to believe that the esophagoscope is almost as dangerous as the blind instruments when passed without any previous practice. Serious and fatal trauma have been caused, so that it is time to issue a warning against violence and carelessness in esophagoscopy, especially by the inexperienced. In one instance, an inexperienced man tried to get out a portion of the cervical vertebrae, believing it to be a foreign body. In another instance, an inexperienced man passed the esophagoscope into the mediastinum. Any man, can, with practice, learn to pass the esophagoscope, and even without practice a careful man would do no harm. I think the points in Dr. Coakley's paper are very well taken and point useful lessons.

Dr. Harmon Smith, of New York: I will report the history of a case of a child who had been sent to me at the hospital and had that morning obtained an X-ray picture showing the presence of a coin in the oesophagus. I entered the oesophagus with a short esophagoscope, but not being able to find the coin,

extended the tube until I had investigated the stomach, where it was still not visible. An X-ray picture was then taken which showed that the coin had passed into the small intestine. The next day an X-ray picture revealed the coin in the sigmoid flexure, and the third day the necessity for an X-ray picture did not obtain. To illustrate what can occupy the laryngeal space and yet be overlooked, I will cite a case of a child who has been recently submitted to my care, where thirty-eight days previously the family physician had been called in, and from the symptoms diagnosed the case as one of membranous croup. Ipecac was given, and the child vomited and appeared better. A few days later it choked up again, and a consultant was called who advised the employment of antitoxin, which was given, and also the introduction of an intubation tube, which was introduced. A culture of the pharynx and nasopharynx was negative for Klebs Loeffler bacilli. The intubation tube remained in forty-eight hours and was coughed up, after which the child seemed to get along very well, though the voice remained hoarse. Later the patient choked again, and he was submitted to a laryngologist, who, upon a cursory examination made with the mirror, thought he determined a papilloma of the larynx. The case was referred to me for removal of the papilloma. With this in view I prepared to remove growths from the larynx and subsequently do a tracheotomy, if necessary, but upon entering the larynx I saw a small glistening object, and with an ordinary pair of forceps removed a closed safety pin.

Dr. Christian Quevli, of Tacoma, by invitation: I had a case in which a child, aged 4, had swallowed a coin. A doctor who was called tried to push it into the stomach, but did not succeed, the coin becoming fixed in the lower part of the esophagus. After administering an anaesthetic, I used an esophagoscope, got hold of the coin, but when it reached the larynx it slipped. While getting ready to go after it again the child died suddenly. I would like to know if it was pressure on the recurrent laryngeal nerve that killed the child so suddenly.

Dr. George A. Leland, of Boston: I wonder how often the old-fashioned methods are used nowadays where there is so much instrumentation. Now, if there is a foreign body in the oesophagus, the first thing done is to run a tube down the throat.

In 1878 or 1880 the captain of one of our United States war vessels in Yokohama harbor swallowed a small tooth plate, which became fixed in the oesophagus. A consultation of four

or five surgeons was held, and oesophagotomy was strongly advised and positively declined. The consulting surgeons thereupon left the vessel, predicting an early demise and bidding a long farewell to their distinguished patient. Soon after their departure his body servant bethought himself of an emetic, and gave him one consisting largely of soft soap. The resulting violent emesis immediately brought forth the offending object, and the captain was all right.

HISTORY OF A BRONCHOLITH, BRONCHIAL CALCULUS OR LUNG STONE.

BY WALTER F. CHAPPELL, M.D.

Laryngologists and rhinologists are frequently consulted for throat coughs and some forms of asthma. For this reason I am wandering into the bronchial region for an opportunity of presenting this unusual case and to show that all causes of asthma are not centred in the nose, nor all throat coughs in the larynx.

Mrs. B., age 52, had consulted me off and on for a number of years for slight sore throats, but nothing of a serious nature. On the 10th of October she was seized with a sudden attack of wheezing, which did not bother her much until the 14th, when it became more severe and was accompanied by spasmodic attacks of coughing, lasting an hour at a time, and by intense tickling in her throat and with little expectoration. Considerable exhaustion followed these attacks.

October 16th my advice was sought. I elicited the history just described and that the tickling was below the larynx and the wheezing in the upper part of the chest in front and behind. No new local conditions in the throat or upper air tract could be found, and I thought the attacks described were due to grapes, which the patient had been eating in considerable quantities. A great deal of mucus was raised during the night of October 19th, and considerable relief was experienced on the 20th and 21st, although the wheezing continued. On the 22d a severe fit of coughing occurred and a small calcarious mass was raised. The intense tickling was relieved at once, but the wheezing continued. October 25th, coughed very hard all night and was exhausted and felt strangely nervous next morning, when suddenly she was seized with a great coughing spell and expectorated the large calcarious piece now shown.

Immediately all wheezing stopped and her breathing became

absolutely free and as normal as it had ever been, and, although much exhausted, a great feeling of comfort and repose followed. No blood or mucus accompanied the expectoration of the broncholith and no unusual chest conditions could be heard and her ordinary state of health was resumed. On February 13, 1913, she coughed up, with slight effort, another small calcareous piece, which was brought to me on April 4th, with the statement that for a week past there had been slight coughing spells, with expectoration of a little mucus and dark specks like blood, chiefly in the morning. A microscopic examination of this sputum showed that it consisted of pus and mucus with some blood cells and superficial epithelium. Staining showed numerous pyogenic cocci and nonpathogenic organisms, but tubercle bacilli could not be found.

The first calculus expectorated did not come to my observation, as Mrs. B. was in the country. That of October 16th and February 13th are both in my possession. The former was the larger and in one piece when expelled. The piece of February 13th was quite small, and when placed by the side of the larger one they joined together and formed a round piece as large as a nickel, slightly concave on one side and convex on the other and the general surface rough and ragged. Chemically it consists of phosphate of lime, as reported by Dr. F. W. Wersebe, of Washington, Conn., who, I understand, examined it.

An X-ray was taken of Mrs. B's chest by Dr. E. W. Caldwell, and is the only one so examined, as far as I can learn. Dr. Caldwell's report is as follows: "I do not find in the X-ray plates of Mrs. B. any direct evidence of a broncholith. There are numerous shadows of small calcarious bodies, but most of these are undoubtedly due to calcified plates in the costal cartilages and to calcified bronchial glands. The lung shadows on both sides are much stronger than normal, and suggest some pulmonary disease. The shadow of the diaphragm on the right side is somewhat irregular, and makes me suspect that there may be adhesions at the base of the right lung. There is a faint suggestion of a cavity in the right lung, but we cannot determine this with certainty."

Mrs. B.'s early life history, as told in her own words, relates

many medical troubles of various kinds, but whether any of them throw light on the present condition of her chest I must leave to your judgment. "When about four weeks old the nurse drugged me with Winslow's Soothing Syrup, and it is a wonder that I live to tell the tale, as the next child she nursed died from an overdose of the same kind of syrup. When twelve years of age I developed a deep hollow cough, which was supposed to come rather from my stomach than the lungs. This was followed soon after by a severe and prolonged attack of indigestion, accompanied by distressed breathing and pain in chest, chiefly the left side, and supposed to be connected with some heart affection. Father died about this time from enlargement of the heart. I enjoyed good health from about the age of fourteen until I was twenty-two, when I had an attack of whooping cough, during the sixth and seventh months of pregnancy with my first child. For several years after I had a great many attacks of neuralgia in my head, and at the age of thirty I had severe intercostal neuralgia, pelvic cellulitis, and a child born at the seventh month. For the following nine years my health was very good, but during the tenth year I had an attack of nervous prostration, and took lessons from an expert in diaphragm breathing in connection with general exercises. Since then my condition has been much stronger and general health very satisfactory until the attack of asthma, which resulted in the expectoration of the calcarious masses I have given you."

The history described by the patient does not in my mind give any definite clue to her having suffered from the disease which we would naturally believe must have preceded the formation of the numerous calcarious deposits, which we know Mrs. B. has in her chest and some of which she has expectorated. The hollow cough and chest symptoms described during her twelfth and thirteenth years may have been an infection of the bronchial glands, which was arrested, became caseous and later calcified, or the same thing may have happened after whooping cough.

Text books on general medicine and pathology just mention the possibility and the presence of bronchial calculi. A care-

ful examination of medical literature reveals few reports of such cases in the English language, but French and German books report more cases and speak more fully of the cause and result of this condition. In conversation with my associates I find some who have seen such cases, but never reported them, and it seems not unlikely that these cases are more numerous than we might suppose. Dr. Walter B. James has given me the following story of an unreported case which occurred in his practice:

Mrs. A., Age 54.

Past History—Weakly at puberty and it was feared she had consumption, but no diagnosis of that nature was made. Later had nervous prostration and, a severe broncho-pneumonia. Eighteen months ago a severe cough began, worse at night and after talking. Some hoarseness off and on; also fever at intervals, at times as high as 101 F. Sputum was blood tinged after severe paroxysms, but no tubercle bacilli were found, although five or six examinations were made.

Present Condition—Appearance healthy, pulse and heart normal, prolonged expiration over both lungs, especially the left. Always feels somewhat oppressed and often puts hand to chest. Cough pretty constant, at times severe paroxysms, face diffused, loses breath, especially when talking or laughing. Two days ago, while reading, severe paroxysmal coughing spells began, accompanied by strangling sensation and expectoration of some blood. After an especially severe coughing spell, which lasted about an hour, spat up a solid mass, which proved to be a broncholith. This was of a yellowish white color, very hard, ragged and irregular, with sharp spicules, and measured one-fourth by one-eighth by three thirty-seconds inches. A bloody fluid with a little grayish mucus accompanied the broncholith, but no tubercle bacilli were present.

The history of this patient was taken ten years ago, and a recent letter to Dr. James states that the cough and bronchial condition, for which he was consulted, ceased with the expulsion of the stone.

A SKULL WITH MALFORMATION OF THE TEMPORAL BONE AND DISTORTION AND ABSORPTION OF THE BASILAR REGION AS IF BY PRESSURE OF A NON-MALIGNANT NASO-PHARYNGEAL TUMOR.

BY ALEX. B. RANDALL, M.D.

The specimen was obtained without history from the Anatomical Laboratory of the University of Pennsylvania and is a brachycephalic skull, without calvaria or mandible, edentulous and apparently that of a well-developed male of about 70 years. It presents quite normal characteristics on the right; but the left mastoid process consists of an irregular group of osteophytes 40 mm. back of which, over the groove of the lateral sinus, is a smoothly bevelled, rounded opening 17 mm. in diameter. No evidences of tympanic caries suggest that the anomalies of the temporal are post-operative. In a patient of such apparent age the smoothing and osteophytic growth would require many years, pushing back the date of any assumed operation to a period when such interventions were rarely if ever so done. The defects here may in all likelihood be set down as developmental.

Study of the cranial base shows loss of the lower wall of the carotid and of the auditory canal, which may be accidental and post-mortem; and much enlargement of the posterior lacerated foramen extending it forward into the middle lacerated and wholly separating the petrous tip from the basilar. This, like the following anomalies, seems absorptive. There is much thinning of the basilar portion of occipital and sphenoid and absorption of the body of the sphenoid almost undermining the vomer at the rostrum. The left sphenoid sinus seems opened by this process; but really its posterior wall has been pressed forward to the ostium, making it a mere cleft. The right has been less interfered with. The left pterygoid process is 8-10 mm. further forward than the right, but there is almost as much displacement of the palate, superior maxilla, malar, and

zygoma on this left side. It is this general thrusting forward of all of these structures on the left with evidences of absorption that speaks so strongly for ascribing the distortion to the pressure of a tumor. There seems no infiltration of the bone as by a malignant growth, and the asymmetry involving the face would point to its effect in early life; so I ascribe the ab-

Fig. 1. Base of skull showing absorption and distortion of palate.

sorption and displacement to an adolescent fibroma of the pharynx-vault.

Study of the interior of the cranium shows little asymmetry except that the left seems to have been the smaller and the anterior and middle fossae accordingly are 6-8 mm. narrower than the right. Absorption has here also played a part—the

basilar process is hollowed out and the posterior clinoid processes almost lost, as if a lobe of the growth had invaded through the foramen lacerum and underlain the pons. In consonance with this the foramen magnum is distorted as though the medulla had been pressed to the right and escaped quite obliquely toward the vertebral canal.

Fig. 2. Interior of cranium showing asymmetry and absorption about the basilar portion and foramen lacerum.

While regretting that the history of this case must be hypothetical and the combination of developmental with pathological alterations open to suspicion, I believe it offers points of sufficient interest to justify its presentation before you.

SCHOLASTIC LAZINESS AND RESPIRATORY GYMNASTICS.

BY MARCEL NATIER, M.D.

All teachers bear witness to this lack of power in attention, to this diminution of energy in application; and doctors, the directors of moral tone of the modern home, attribute it to overwork or to misdirection of effort.

(*H. PARIGOT, "The Crisis of French."*)

Laziness, generally, is a sin; moreover, as taught in the catechism, it is a mortal sin.

Is *Scholar Laziness,* because of its nature, sometimes susceptible of remission?

Being in full possession of their entire sensorial faculties, many children do not use and practice them, and by the lack of attention they do not interpret the sensation they receive by reflection. Then, the difference between their development is exactly explained by the degree of attention of which the intellect is capable. The progress of comprehension is, in fact, derived from attention which permits comparison, judgment and memory.

Certain children are naturally attentive and the remainder is the exception. The majority, in an early period, show themselves diverted. We must call their attention, which is obtained with difficulty, or sometimes never, to captivate after having them awakened. Whence the scholars qualified of laziness increase the thick battalions of "Cancres."

In the *Imaginary Patient,* Diafoirus, taking his son as a model, gives the following and sufficiently exact description of "Cancre":

"He never had a live imagination, neither the flame of spirit that is noted in some children . . . When he was small, he never had been what is called smart and sprightly . . . One had all the trouble of the world to teach him to read, and at nine years of age he did not recognize the letters . . ."

Looking without seeing, listening without understanding, "Canere" is really a simply passive being. His inattention alone prevents him to assimilate. He cares very little about the changes brought to the programs of lessons. He will always know how to escape from intellectual overwork.

Scholastic laziness is often due to fatigue, which is connected itself with lack of normal breathing.

Following is the way it works:

The incomplete development of the thorax provokes circulatory troubles, followed, in the long run, by general anemia with circulatory disturbances of various organisms, mainly of the brain. The retention of the waste of cerebral substance, favoring the intoxication with a consecutive exhaustion of the gray cells opposes, gradually, intellectual effort. Consequently "canere" could really be considered as a pathologic individuality. Thence it is allowed to ascertain beforehand—at least in certain cases—the efficient role possible of the doctor in the treatment of Scholastic Laziness.

Remarks: Boy 8½ years of age. Father very intellectual; extremely nervous; dyspectic. Mother less nervous; generally good health.

Previous Personal State: Birth in good condition. Raised at home with good care. Normal growth.

At the age of 5 years remarkable breathing; uneasiness. Ablation of adenoid vegetation in November. Later, during three months, frequent serious anginas; injection of antidiphtheritic serum. In May next, measles, weak; in July, serious scarlatina.

Actual State (Oct. 25, 1906): Height over the average. Physiognomy awakened. Face emaciated; complexion sunburnt. Skin (dull) rough at touch, dry and thin. Members rather small; less developed on the right than on the left. Frame bony, rather strong.

Respiratory System: Anemia of the pituitaire. Nose dry. Complete permeability of superior air passages. Slight paresis of larynx with hoarseness.

Pointed shoulders; clavicles, ribs and shoulder blades pro-

ject; sternal depression is noticed. Asymmetry of bust because of arrest of development of the right side. Cervico-dorsal scoliosis at the left convexity. Lumbar saddle back; Sacral-fossa reduced in size.

Respiratory movements rudimental; almost complete silence of high notes.

Digestive System: Appetite mediocre; frequent indigestion with vomiting. From childhood up to this minute: obstinate constipation—regular use of calomel.

Urinary System: Urine not abundant and heavy.

Treatment: Regular and methodical exercise of respiratory gymnastics.

Result: Attested already objectively by the comparison of figures.

They consist at the end of the treatment, in:

(a) Growth of height (0 m. 013);

(b) Increase of weight (4 kil. 870);

(c) Extension of chest (0 m. 06 cent.);

In proportion of a child of the same age, the patient indicated, then, a superiority of 6½ cent. for the height, and 8 kil. for the weight.

Notice also; the recovery of appetite; disappearance of indigestion and of constipation; the regularity of urination; the freshness of complexion; the whiteness, smoothness (softness) and thickening of the skin; the almost complete symmetry of thorax; normal amplitude of respiration.

But a capital point that will retain our attention—the change has been chiefly observable is intellectual and mental.

The following annotation, written by the father, will permit judgment of it:

After a somewhat long walk the child appeared in the evening with his features drawn and heavy rings under his eyes, without any appetite and not wishing to do anything but sleep. It was the same after some hours of play with his comrades.

When I entrusted my son to your care, he had just finished the eighth grade in school.

Of an apparently active intelligence, rational in the ordinary

acts of his existence he seemed incapable of the slightest mental strain or of any effort at attention. It had taken two years to teach him to read and success at that had not been very marked. He was seven years old. His spelling was most weird; he never applied even the elementary rules of grammar, which he knew well enough, and the greatest errors, having been pointed out to him, rendered him confused for an instant only to be repeated over again a few minutes later.

Upon beginning his eighth year the child returned pale and thin from Switzerland, where he had spent two months. He comported himself rather well during his eighth year, but at the end he seemed tired and it was high time that his scholastic work cease. Two weeks passed at Trouville greatly aggravated the fatigue which he had not entirely recovered from during his sojourn in the country, where he spent the rest of his holidays.

Concerned over that intellectual ineritia, attributing it to a carelessness for which the child seemed to me culpable, worried about the future, I had punishments follow reprimands, without arriving at any more satisfying result.

His beginning in the seventh year class was so wretched that I did not hesitate to take him out at the end of three weeks. It was then that I put him in your hands.

All intellectual work was suspended during the four months of treatment. The school year was so far gone at the end of the cure that it hardly seemed worth while to utilize the few remaining months.

During his vacation, however, I had him do some exercises. The first dictation, of a dozen lines, contained some thirty odd errors; that was no better than in the past. But the second already showed progress. Genders and numbers agreed. At the third participles no longer ignored their complements, and at the end of about two weeks the errors were limited to unfamiliar words of which his memory had not retained the exact form.

In the beginning of October, 1907, the seventh year, hardly begun the year preceding, was taken up again. From the start the pupil ranked among the class leaders, and with the ex-

ception of some inequalities at the beginning of the Summer he kept his rank.

The sixth year has been excellent; and actually in the fifth year of high school he holds among thirty-two scholars first place. His lowest rank was that of third in Latin translation. His marks are good; his professors whom I have seen consider him as markedly intelligent, reasoning well and with the ability to form his own deductions. His tasks are correct, his Latin exercises, composition work of observation, which in the beginning above all are a work of observation, contain few faults.

The attention is of the kind that with difficulty resists the roguery of some playmate, and which without any doubt is more captivated by the adventures of Sherlock Holmes than by those of the first kings of Rome. That, none the less, is a mental state quite satisfactory in a child of twelve, who has entered the normal school well prepared both physically and mentally.

You know how very grateful I am to you

Later, general development very regular. This year (1913) in the second class, the child gave full satisfaction both to his parents and teachers.

The theory of nasal aprosexy according to which adenoid vegetations, especially, would have been one of frequent causes of scholar laziness bears, in its kind, a formal contradiction.

In addition to it and by a strange contradiction, the adenotomy seemed, on the contrary, to involve the balance of the intellectual inactivity witnessed later on the little patient.

To record—important fact in my opinion—the part played by the consecutive insufficient to the inco-ordination of various segments of the respiratory tree considered as the whole. The only correction of the respiration, in fact, is sufficient to re-establish the organic rhythm deeply disturbed. In such cases, the circulation is regularized; the equilibrium recovered and maintained by the internal and external secretory glands, warrants and assures the normal work of exchange and elimination. Thus the general nutrition improved, one may see, if it

is still time, to share in the common benefit, the brain whose operations are, every day, becoming easier, brighter and more precise.

Resume.

The laziness of scholars occurs usually from fatigue. This latter engenders, in its turn, the inattention that transforms progressively a good scholar to "Cancre."

The adenoid vegetations only are powerless to explain, as it is most generally admitted, the birth of the majority of scholar laziness cases. Based on the example mentioned here —besides not exceptional at all—the contrary thesis has nothing improbable or excessive. It is presumed that, from a respiratory insufficiency point of view, the etiologic basis of this variety of laziness ought to be remarkably enlarged.

With suitable exercises, one can conquer, logically, the fatigue and also its consequence that is laziness, when one as the other is certainly caused by respiratory insufficiency.

Laziness, on the contrary, connected with irremediable physiologic deficiency, the real true reason has to be explained to the parents that have to yield to evidence and know, according to the precept of Montaigne, "rank their desire to the power" of their children.

Conclusion.

There always will be, whatever the parents, physicians and schoolmasters do, some lazy scholars.

Intelligence is a faculty; one might cultivate it; it is useless to pretend to endow him, artifically, who is deprived of it. But great chances of improvement are occasionally reserved for lazy scholars, when the origin of their defect is recognized, one knows how to remedy it properly. *"Mens sana in corpore sano."*

The doctor, called for this purpose, will look, by a careful examination to find out if the child possesses really the necessary physical qualities in order to start, with hope, the intellectual operations necessitated by correctly conducted studies. The teacher, too, having charge to manage a pupil in good health, possessing perfect equilibrium, will be able, with-

out overwork or maltreatment, to obtain the maximum of result.

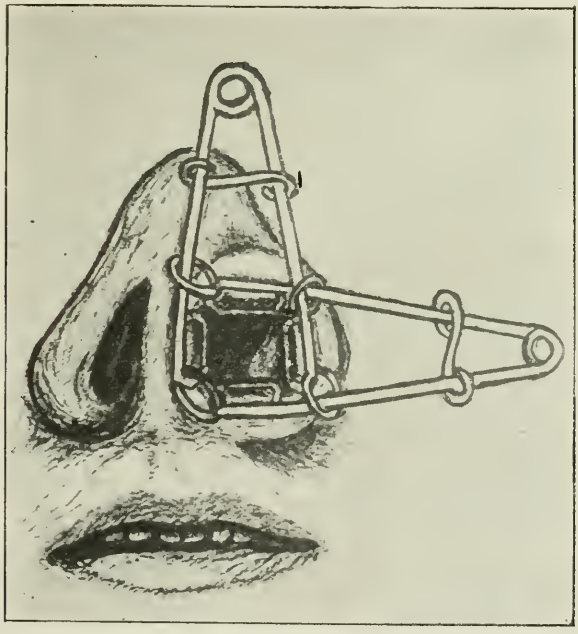
Boarding schools are, as much as possible, to be avoided for the lazy scholars attacked by respiratory insufficiency. It is appropriate, in fact, for such children to experience the advantages of outdoor life in a purer air and more abundantly oxygenated. For the very same reasons they should not be deprived of recreations and of going out. It is also well to compel them not to remain a long time in a bended position on the desk, because these are deplorable proceedings that exaggerate circulatory and respiratory disturbances already very evident.

Contributing willingness, by this competition of making good, will result, finally, for the child in a positive gain; parents, physicians and teachers will all be authorized to show themselves legitimately proud.

PRESENTATION OF INSTRUMENT—A SELF-RETAINING NASAL SPECULUM.

BY DR. WILLIAM E. CASSELBERRY.

The self-retaining nasal speculum, first presented three years ago, is changed in a manner to render it bilaterally applicable without disconnection for rearrangement of its two pairs of blades, which are now permanently looped together, thus greatly facilitating its use. By expanding the nostril in both directions it not only provides the maximum exposure without material discomfort, but holds itself in position so securely that the line of view can be slanted in any direction by leverage without causing the instrument to slip out. (Fig. 3.)



Self-retaining nasal speculum.

Candidate's Thesis.

AFFECTIONS OF THE NOSE, PHARYNX AND LARYNX, DEPENDENT UPON ENTEROGENOUS AND AUTO-INTOXICATIONS.

BY RALPH BUTLER, M.D.

Twenty-five years ago Beverley Robinson, in an address delivered before this Society, made the following statement, "Let the stomach perform its work well, and the nose, pharynx and larynx are not apt to be great sufferers from catarrhal inflammations."

To the present-day student of rhinolaryngology the above frequently quoted statement is first read with a sensation of surprise, for the more definite subjects of surgery, bacteriology and reflexneuroses lend themselves more readily to the study of our specialty than do researches on the complexities of metabolism, including the chemistry of the secretions and excretions. Neither does the stomach present itself to the internist as an all-important organ, but there is an underlying thought in the quotation that demands attention.

The improved methods of surgery and the favorable results obtained thereby in nose and throat conditions have claimed so much attention that the general health of the patient has been insufficiently considered. In searching for etiological factors in diseases of the upper respiratory tract, attention is given first to inflammations, obstructions, adenoids, tonsils, etc., not forgetting the importance of free ventilation of the attic and the drainage of the sinuses.

When there is a reasonable probability that a definite local lesion is the cause, or one of the causes, of the symptoms, it should be removed, but it is unreasonable to expect surgery to be wholly successful where there is an underlying constitu-

tional disturbance partially responsible. Unless this is borne in mind there will be many a straightened septum, operated sinus or tonsillectomy that will not give the desired relief. Dr. Abraham Jacobi once well said that there was many an ovary and many a turbinal in alcohol that should be in its place in the human body.

In order that mistakes may be avoided in the selection of cases for operation, it is essential that all the details of the general condition be carefully considered. One celebrated author contributes a valuable treatise on the nasal accessory sinuses, and not once does he mention the lowered vitality of the patient as a predisposing cause of sinus disease. A physician of my acquaintance suffered two operations on the maxillary sinus via the canine fossa. Both were performed by prominent rhinologists, one in this country, the other in Europe. He finally consulted the late Professor Fränkel of Berlin in regard to the unhealed sinus, fully expecting a third operation with the usual suffering. To his surprise, the kindly Fränkel told him that an operation was not necessary, and advised him to work less, to eat and sleep more, and to build up his general health. The patient recovered quickly and had no recurrence for sixteen years, and then only a slight one caused by overwork and worry. He has a septum deviated markedly to the side of the affected sinus. It is with these actual and the potential failures that we wish to deal in this paper.

In the majority of text books on rhinology many pages are devoted to surgery and topical applications, and so little space to the general condition, that the importance of the latter is overlooked, and the inexperienced subject patients to operations and long periods of local treatment with inadequate improvement. The tendency is to operate and to treat locally rather than to study obscure systemic conditions, in spite of the axiom that the cause must be removed to prevent the continuation of a morbid process.

Some ten years ago I treated a medical student who was thought to be suffering from a moderate atrophic rhinitis, secondary to a chronic ethmoiditis. He was treated for years by

specialists and was seen in consultation by some of our best rhinologists. Many remedies were used locally with very little benefit. His bowels were regular and his general health seemed to be good. He has recovered from his nasal trouble, which he believes was due to digestive disturbances he now realizes he had at that time.

The fact that local disturbances are often but symptoms of constitutional affections cannot be too strongly emphasized, nor too often repeated. A study of literature pertinent to this subject and daily clinical observations afford convincing evidence that some of the acute and chronic affections of the upper respiratory tract are due to the products of gastro-intestinal decomposition. Guelpa, whose writings on auto-intoxication have created so much interest in Paris, concludes that the essential factor in many chronic diseases is a condition of auto-intoxication. Ziegler writes: "There is no question but that the ingestion of sweets, tea, coffee and other articles of that character will create a disturbance that is first manifested in the stomach, from there is transmitted to the nose and thence through the tear duct to the eye." Bassler says that more or less pharyngitis is commonly seen in digestive disturbances. French claims that in a very large majority of cases of chronic catarrhal pharyngitis the cause of the disease will be found in some portion of the gastro-intestinal tract. Ballenger believes there is a close relationship in many cases between pharyngeal disease and the hyperesthetic types of rhinitis and diseases of the gastro-intestinal tract. Combe in his book on auto-intoxication says: "The frequent and repeated congestions of the throat, pharynx and tonsils in their recurrent forms have been so often noted in our observations that we feel called upon to mention them, for the recurrent anginas constitute a noteworthy symptom of intestinal auto-intoxication." Grayson maintains that without chronic auto-intoxication the neurotic nose would be unknown. It is common knowledge that acute pharyngitis or rhinitis, caused by indiscretions of diet or the ingestion of certain foods, such as strawberries, may be relieved by a purge.

Both the skin and the respiratory tract eliminate waste ma-

terial. The latter eliminates carbonic acid, ammonia, acetone and probably other metabolic products. Both are affected in such conditions as urticaria and pruritis. The skin lesions in these affections are admittedly due in some instances to irritants derived from the gastrointestinal tract and it is reasonable to suppose that the lesions of the mucous membrane have the same origin. The writer has frequently observed granular pharyngitis associated with acne. Other substances are eliminated by the salivary glands, and Kyle maintains that the nasal and salivary secretions become irritating as a consequence of faulty digestion and faulty elimination from the intestinal tract. Further, it is only necessary to note the inflammation of the upper lip in acute coryza to realize that the nasal secretion may be exceedingly irritating. An external irritant long continued may produce rhinitis. The lactic, acetic and other acids produced in the intestines by the saccharolytic and amylolytic bacteria are irritating if brought into contact with the nasal mucous membrane, and the products of the proteolytes may be. It is probable that the absorption of these acids reduces the alkalinity of the blood and thus produces an irrition of the mucous membrane under discussion.

De Schweinitz quotes Horner, Sachs, Förster and Groenow and Elschnig as supporting the view that alcohol-tobacco amblyopia may be due to gastrointestinal disturbances produced by these drugs. He and Edsall found an excessive excretion of enterogenous decomposition products in the urine and a more or less marked urobilinuria in all the cases of the series studied. These anomalies nearly or quite disappeared under treatment coincidentally with improvement in the eye conditions. They urge that these facts give just ground for the belief that toxic substances produced in the digestive tract or in the course of metabolic processes have at least a part in the production of amblyopia in these cases. Other oculists present much valuable evidence to substantiate the opinion that various eye lesions are caused by toxins from the gastro-intestinal tract. If the eye is affected by the gastro-intestinal disturbances and by enterogenous toxins produced by these drugs, as well as by the drugs themselves, it is even more ra-

tional to believe that the respiratory system, by reason of its eliminative function, is also affected by these poisons.

In many of the cases under discussion the severity of the subjective symptoms is out of all proportion to the local changes. A common complaint is an obstinate, chronic dry cough, with negative findings in the thorax, or there may be severe pain on swallowing, without apparent change in the throat. There may be sneezing, with or without a watery anterior nasal discharge from an apparently normal nose. On the other hand, the objective symptoms may be the more pronounced. Frequently we find a dull red or beefy swollen mucous membrane more or less uniformly distributed from the tip of the nose to the bifurcation of the trachea. Occasionally only the larynx, the trachea or the nose is involved, but the mucous membrane of the pharynx is the one most frequently affected. Usually the mucous membrane is a darker red in these cases than in the direct infections, but it may be pale in a vasomotor disturbance, caused by the action of enterogenous toxins on the sympathetic nervous system.

At times the general appearance of the patient suggests the origin of the disease, e. g., a corpulent, plethoric, short-winded individual whose mucous membranes are of the beefy red type already mentioned, is probably a victim of toxins. There may be present such conditions as a muddy complexion, dull headache, lassitude, coated tongue, fetid breath, drowsiness in spite of sufficient hours in bed, insomnia or heavy, unrefreshing sleep, fatigue on slight exertion, lack of mental or physical energy, and constipation or lienteric diarrhea, all symptoms of auto-intoxication. The fact that the bowels move freely and regularly does not prove that the intestines are free from excessive fermentation and irritating toxins. The patient will frequently give a history of excessive, indiscriminate or rapid eating, with insufficient water drinking, little exercise, attacks of hoarseness, frequent "colds in the head," with an improvement of all symptoms in warm weather or after a few weeks on the football field or in the crew.

In establishing a diagnosis the aid of an internist and a physiological chemist should be secured. The internist should

study thoroughly the condition of the organs, viscera and nervous system, for a gastro-intestinal disturbance may be secondary to other acute or chronic diseases. Evidence obtained by palpitation of fecal accumulations in the colon, or the results of colon flushing after stool, is of importance. Indicanuria, especially when persistent, is perhaps our most valuable laboratory finding, but its absence does not prove that gastro-intestinal intoxication is not present, and it may be caused by many other conditions, e. g., bronchitis, bronchiectasis, pus, anemia and possibly temporary constipation. Fatigue toxins, as well as those produced by impaired functions of the ductless glands, may produce symptoms that cannot be positively differentiated from the results of intestinal intoxication.

In a series of cases in which the writer had thorough laboratory and physical examinations made by Dr. C. A. Fife, the findings were valuable from a clinical standpoint, but were not of sufficient uniformity to enable us to draw definite conclusions. Frequently no indicanuria was found in cases which, from clinical observations, were undoubtedly due to enterogenous intoxication. In a number of cases an excessive fermentation of the stools was the notable finding. In all, the improvement under treatment was satisfactory. Until the physiological chemist is able to give us more definite information, we must depend largely upon the clinical signs, at the same time continuing the study of the urine, stools, blood, etc., in the hope of gaining more accurate knowledge of toxic substances.

These patients require local and general treatment, in many instances the latter being the more important. Undoubtedly a badly deflected nasal septum or an offending turbinal should be treated surgically. The improvement in nutrition following adenoid operations indicates that nasal obstruction interferes markedly with oxidation. And it has been demonstrated by experiment on young animals that the partial closure of the nose retards development. Furthermore, the reflex pain and irritation caused by intra-nasal pressure and obstruction may, by their general depressing effect, interfere with metabolism and establish a vicious circle by causing indigestion in much

the same manner as do worry and certain emotions. A diseased tonsil or a suppurating sinus increases the total quantity of toxins to be removed by the already overwhelmed organs of elimination and may be sufficient to break the compensation.

Local applications to an irritated mucous membrane are soothing, if not curative, and astringents to an inflamed pharynx are often of decided value. Most of our patients have such confidence in topical applications that they should be employed if only as a placebo while we are studying the underlying etiologic factors.

The general treatment, as well as the diagnosis, should be under the control of the internist, for although occasionally the simplest directions in regard to exercise, diet and laxatives are sufficient to relieve the patient, frequently it will tax the resources of the internist to discover the cause and to see that the patient complies with his instructions; but it is desirable that the rhinologist should understand the medical as well as the surgical phase of his cases, lest he lapse into the state of a simple mechanic. As a rule, the specialist appreciates more fully than the practitioner the relationship between the diseases of the upper respiratory tract and the general system.

The proper diet can be best determined by studying the results of a test-meal and by examining the stools, urine, blood, etc. The excessive use of proteids, fats and sugars is especially objectionable. Among the many millions of micro-organisms which are found normally in the small and the large intestines, we are particularly interested in two groups, the saccharolytic or amylolytic, and the proteolytic. The organisms belonging to the former are found principally in the small intestine and feed on sugar, dextrine, starch and hydrocarbons, and produce acetic, lactic, succinic and butyric acids. These acids are irritating but not essentially poisonous. The proteolytic organisms are found in the colon, feed upon nitrogenous substances and eventually produce toxins or putrefaction which are deadly. Metchnikoff showed this difference in toxicity by placing minced meat and water in one tube and minced vegetable and water in another and inoculating each with fecal matter. After two days a part of the contents of each was injected

into rabbits; the former produced death, while the latter appeared to be harmless. It has been found that the saccharolytes inhibit the development of the proteolytes, and that the latter thrive only in a distinctly alkaline medium. Herter and Kendell found that monkeys and cats when fed on protein food exclusively produced proteolytic bacteria, but if fed on carbohydrates only non-proteolytic organisms were found, with a reduction of indican and aromatic oxyacids in the urine, and indol, skatol, phenol and bound sulphuretted hydrogen in the feces. It is for these reasons that the reduction of proteid foods is suggested.

Guelpa in his extensive writings on auto-intoxication reports remarkably good results by periods of fasting and purging, his object being to burn up waste materials and to remove the products of this combustion freely and regularly by purging. Arnold in his translation of Guelpa's book says: "If one tithe of his claims for disintoxication treatment can be substantiated, neglect of this method is much to be deplored." I have seen it employed in two cases, in both of which the improvement was striking and immediate.

Intestinal antiseptics and colon flushings are valuable, but do not remove the cause nor prevent recurrence. The drinking of plenty of water between meals aids elimination, that of the skin being increased by balneotherapy. Physical exercise, especially for the patient of sedentary habits, is, in the experience of the writer, one of the most valuable therapeutic agents. It increases the force and rapidity of the circulation, thus improving its efficiency in transporting the waste products to the organs of elimination; it increases the oxidation of effete materials and the activity of the glands in general, especially those of the skin; it improves digestion and tends to relieve constipation. Naturally, exercise in the open air is more efficient. When an athlete abandons his exercises for a sedentary life, these conditions so frequently develop, to disappear again after resuming exercises, that I am inclined to believe in the saying: "Once an athlete always an athlete or a melancholic." French quotes Mulhall in regard to pugilists as follows: "Most of them, as a consequence of a bar-room life, cheap cigars, over-eating,

vast quantities of alcohol and inactive life, suffer from chronic catarrh. If you will but take occasion to examine one when he has trained for a fight you will find that his naso-pharyngeal symptoms have disappeared; he no longer hawks and spits and suffers from stuffy nostrils. He has, through training, eliminated all obnoxious material from the body and secured perfect vascular equilibrium.'' In cases complicated by fatigue toxins, change of scene with relaxation from worry and overwork is of the greatest benefit.

The study of metabolic processes is so complex, and the various factors so interwoven, that it may require a deal of good judgment to produce a cure. It is the more important, therefore, that this phase of our specialty should have greater attention and each case should be studied medically with the hope of obtaining as nearly as possible an exact diagnosis.

The advanced internist is looking more and more to the subject of auto-intoxications as a factor in the explanation of many chronic diseases, and it is for this reason that he sends his cases to the rhinologist for the exclusion of infected tonsils and sinuses as a source of systemic poisoning.

In conclusion, it must be recognized that the subject under discussion does not lend itself to exact deductions, for while it is well known that enterogenous toxemias and auto-intoxications exist, very little is known of their exact nature. Until the physiological chemist discovers the entity of these various toxins the pathogenesis cannot be put on a scientific basis.

Candidate's Thesis.

EPIDERMOLYSIS BULLOSA HEREDITARIA OF THE MUCOSA.

BY ROCKWELL A. COFFIN, M.D.

In reviewing the literature on this subject the writer is convinced that more time should be given by laryngologists to the study of some of the rarer diseases of the mucous membranes which also attack the skin, such as epidermolysis and chronic pemphigus. A correct diagnosis should be made early in these cases, as the prognosis is so entirely different in each instance. The literature gives only fifty-two articles on the former disease which the author has been able to find to date, consequently we must consider it one of the rarest diseases attacking the mucosa.

In 1882 Goldscheider first described the symptoms, and in 1886 Kobner first designated epidermolysis as a distinct disease. The direct cause has not yet been demonstrated. Sections of skin made by Engman and Mook show oedema of the epidermis, succulent horny layer, normal granular layer and dilatation of the intercellular canals, no marked degeneration of the epidermis, and only changes that could be ascribed to oedema. Oedema of the cutis, dilatation of the lymphatic channels and vessels most marked in the upper portion, papillary pigments therefore swollen, slight increase of cells about vessels, absence of elastic tissue in the papillary and sub-papillary layers of the derma and sparsely distributed and deformed in the deeper layers. Sections of bullae show a lifting up of the entire epidermis from the derma. A flattened line of cells on the floor of the bleb seems to be the cause of resisting the flow of serum. The elastic tissue is absent in the bullous as well as the normal skin of people affected with this disease.

Sutton found in four cases with monthly examinations an average of 9,600 white blood count and 37 per cent polymorphonuclear and transitionals.

A typical case which came to the throat department of the

Boston City Hospital will well illustrate the symptoms of the disease.

M. B., aged 37 years; a native of Alsace; painter. Family history negative. Presented himself for the relief of pain in the mouth and difficulty of deglutition. General nutrition poor, and urine was found negative. Examination of the mouth showed along the edges of the teeth many superficial sloughs on lips, gums and cheeks, and some small bullae varying in size from that of a pea to a quarter dollar. Some of the lesions were covered with loose mucous membrane, evidently of recent occurrence, while others were reddish and covered with moist secretion. Others extended from the mucosa well over on the true skin . The mouth condition had existed more or less ever since he could remember. Recently he has been drinking hot brandy, and since then has experienced a burning feeling extending well down into the stomach. Can eat only liquid and semi-liquid food.

The pharynx examination showed lesions similar to those in the mouth, but not so well defined. Epiglottis and arytenoids covered with a superficial slough, evidently the remains of old bullae. Dentition defective, he having only five lower teeth. Bullae along both true and false teeth.

As a consideration of the skin lesions is most important in making a differential diagnosis from chronic pemphigus, a description is most necessary, as the lesions were typical of those given in most of the cases reported by others. With the exception of two undeveloped finger-nails on the left hand and one on the right, there has been an entire absence of finger and toe-nails since birth. On the body a pemphigoid eruption extending over the abdomen and limbs, of a vesicular blebby character. All lesions occurred at points of irritation or trauma. No limit to size. Extensive seborrhoea of scalp, with much itching, but no itching of the body. Palms of hands and soles of feet affected at times. While demonstrating the case to students, one of them in holding out the tongue used a good deal of pressure, and, true to the prediction of the patient, a bleb formed at the point of pressure. After a contusion there is a reddening of the part, which gradually fades, and on the

spot a bleb appears in a short time, which is filled with a light serous fluid, sometimes hemorrhagic, and at times, if it becomes infected, pus forms. As there were no mouth lesions mentioned in many of the reported cases, we must take it for granted they do not always exist. Morris reports having seen casts of the esophagus, and it is quite likely this case also had esophageal lesions on account of the sensation of burning which he experienced. A little more than half the cases were congenital rather than hereditary. Several cases had their beginning between twenty-four and forty-five years of age, although nearly all began either at birth or during the first year of life. The symptoms of the inherited cases began, as a rule, earlier than the congenital.

Disappearance of symptoms occurred in several cases later in life. Intercurrent diseases have no effect on the amount or kind of fluid in the blebs or cause of the disease. Berger shows no formation of blebs by application of chemical agents or heat, but X-rays will cause blebs to form. Pernet describes a similar condition in the eyes, but as it is not mentioned by other authors it must be exceedingly rare. Engman claims by grasping any part of the body between the thumb and index finger, and at the same time using severe traction, the epidermic layer will suddenly give way and slip off the underlying tissue in the shape of a figure eight, leaving an oozing surface similar to that left by the bullae. It did not separate in four cases of pemphigus on which it was tried .

Many cases of epidermolysis complain of slight chilliness. Because of the rarity of both epidermolysis and chronic pemphigus, and the identical microscopical appearance of the lesions, it is most necessary to differentiate between the two before considering any other diseases. One must bear in mind the possibility of pemphigus lesions appearing at some point of trauma, but careful examination will always reveal lesions which are not accounted for in this way. Pemphigus appears, as a rule, later in life, while epidermolysis appears practically always either at birth or within the first year after. Therefore, the time of its beginning and the location of the lesions are the principal things we must consider in differentiating these two.

The elevating test (firm pressure on the tongue) will also aid in determining with which we have to deal. There will be a negative result in case of pemphigus. Absence of finger and toe-nails from birth is very suggestive that it is one of epidermolysis. Involvement of the conjunctiva is common in pemphigus, while only one case with this symptom has been reported of epidermolysis. It must be borne in mind that several cases have been reported—including one by the author—where lesions of pemphigus have existed in the mouth for years before the skin or conjunctiva have become involved. This does not happen in epidermolysis.

Syphilis should be ruled out in this disease, as in practically all diseases of the mucosa. Since the use of the Wasserman and other reactions of the blood have proven so helpful in most cases it is quite easy to eliminate syphilis, especially as bleb formation in lesions of the mouth is not at all characteristic.

Herpetic blebs of the mucosa are more regular in shape and inclined to have a reddened areola at some stage of development, and are not produced by trauma, but appear along the course of certain nerves.

One might say the prognosis is favorable as far as life is concerned, but the prospect of cure is practically impossible at the present time, although one or two cases have disappeared later in life.

Many things in the pharmacopoeia have been tried for treatment first and last without benefit, with the exception of arsenic, which in this case responded slightly at times to Fowler's solution, together with removing all sources of irritation and trauma, and keeping the patient in a temperature as low as can be comfortably borne.

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Candidate's Thesis.

IMMEDIATE AND REMOTE SEQUELAE OF RADICAL REMOVAL OF THE FAUCIAL TONSIL—OBSERVATION OF TWO HUNDRED CASES.

BY VIRGINIUS DABNEY, M.D.

When we reflect that operation looking to the removal or reduction in size of the tonsil was devised and practised in the middle ages, the amount of literature on this subject in all its phases is in no wise surprising, great though it be. Hence, a few only of those articles pertinent to the limited scope of this paper will be quoted here. Similarly, the facility with which any point of view could be sustained by reference to these authors is obvious, but it is one thing to state a fact and quite another to account scientifically for its existence. Thus, the statement of sterility in the male or female who has had a tonsillectomy done before puberty, the occurrence of blindness and other calamities of physical origin, all may have existed but such *post hoc, propter hoc* reasoning cannot convict the laryngologist.

In order to narrow the field of this paper the radical removal alone is considered, which can, of course, be done by as many different instruments as methods: obviously, again, the partial and the radical method may share in common some of the sequelae, notably hemorrhage and injury to the adnexa. Consequently, I do not wish to be understood as limiting any given result to the radical method merely because I mention it in that connection.

The agitation to stay the hand of the laryngologist in his alleged ruthless onslaught on the innocent tonsil is timely, if a little overdone, in so far as it has directed attention to the harmful effects that may and do follow the removal of these problematic organs. This plea for considerate surgery is eminently proper, but the lay press has made such spectacular use of it that a legitimate operation is now performed under great

protest on the part of parents who have had their opinions moulded by the public prints, even where it is vital to the patient's health.

Primary and Secondary Hemorrhage.—This sequelae is given prime consideration by all observers, though a few seem to regard it as not especially serious (Freer, Richardson, Ballenger), from which view I must sharply dissent. Although I have never had nor seen a fatal hemorrhage following an operation on the tonsils, yet I have been profoundly alarmed at several of my own cases, and have seen some that resisted the skill and dexterity of experienced operators. The sinister and disconcerting fact is, in my mind, that patients have died from this cause. A colleague told me of a fatal case he had had in which the patient, a young girl of about 15 years of age, swallowed quietly the steady ooze from the tonsillar cavity till exsanguinated. Her pallor finally attracted the attention of a nurse, who gave the alarm, but it was too late. This case had had nothing approximating a hemorrhage at operation, and there was no reason to fear any complication, though she really died of neglect on the part of the ward nurse, who should have watched her carefully.

E. A. Crockett, in stating that he considers hemorrhage a serious matter, says that in a year and a half 12 deaths from this have been reported in and about Boston, leading him to infer that there are many more than that number. However, in his discussion he brings out the fact that only one in every group of twelve untoward sequelae of the tonsil operation was noted as occurring at the hands of a competent operator, meaning either a laryngologist or an operator who had had experience and training in this work. In addition to the twelve deaths referred to, he records "many serious hemorrhages," one requiring ligature of the common carotid after a scissors enucleation, and two deaths in two minutes following a sharp dissection with the scalpel—truly an astounding calamity, absolutely inexcusable. None of these cases was his own, though he lost one patient from ether pneumonia followed by empyema, acute mastoiditis and meningitis. Lindley Sewell was able to find reports of nineteen deaths and fifty severe

hemorrhages which were controlled. It is his opinion that the menstrual period conduces to greater loss of blood if the patient be operated upon at that time. I have not been able to observe any difference, though I have repeatedly operated at this period and made careful notes on every case in order to determine this point. Ballenger, in 9,000 cases, had only two hemorrhages severe enough to cause marked anaemia and exhaustion. Pfingst observed a haematoma in the pharynx as a result of suturing the pillars over a pledget of gauze to control a hemorrhage which began eight hours after operation. Cassellberry thinks that about one case in every seventy-five caused him some difficulty in stopping the flow of blood, out of an experience represented by 1,500 cases. One case which caused me much anxiety was the result of a punch operation, coming on three hours after he had left my office. The flow was controlled with lumps of ice fitted snugly into the bleeding cavity. Ten days later while walking slowly in the street his throat began bleeding and did not stop till he had fallen from syncope. I have seen twice in consultation an apparently uncontrollable primary hemorrhage due to failure to clean out thoroughly the supratonsillar fossa. A hard, fibrous nodule containing bleeding vessels was in each case discovered, and its snaring immediately stopped the flow of blood. I have observed that there are three points whence stubborn bleeding may arise in the radical operation: the supratonsillar fossa, the middle third of the posterior pillar and the point where the infratonsillar fossa blends with the pharyngeal wall. In the method which involves divulsion of the loosened and hanging tonsil there is apt to be a stripping of the small vessels in their length, which will always cause a type of bleeding necessarily and, for obvious mechanical reasons, stubborn. I have had three cases which required suturing of the pillars to control secondary hemorrhage, and innumerable ones in which I deemed it wiser to crush bleeding points or areas with powerful forceps rather than to try pressure, though the latter method would have in time controlled the trouble, I feel sure. Stucky reports a death a few hours after operation from exsanguination, and nine cases of secondary hemorrhage which required

a second anaesthesia and ligation or suturing of the pillars. He does not give the names of the operators, or any particulars of the methods employed at operation. C. W. Richardson in a very frank and comprehensive paper on complications of the tonsil operations reports no serious bleeding in his own cases, which he attributes to his technique of finger enucleation. In this I think he is correct, though I do only a partial finger separation and finish with the snare.

Hyperpyrexia.—Richardson and Wishart both report a fatal case with this complication, in which the temperature rose to 107 F. The former case had a temperature of 99.4 F. at operation. To be classed under this head, perhaps, is the case of a chauffeur of good habits whose tonsils I removed under the severe handicap of a badly given anaesthetic. He was never sufficiently relaxed to dispense with the orderly's help, and the hemorrhage, from two torn vessels on the posterior pillar, rendered my task particularly difficult throughout. From normal at operation his temperature in two hours rose to 102 F., and in four hours to 105 F., and he was in a mild delirium for twenty-four hours. His chest, urine, blood and history were all negative. By lysis in five days his temperature became normal, but it was a month before he regained his strength, though an unusually muscular man. All temperature readings were by axilla, which would show more fever than indicated by the figures 105 F., of course.

Infarct of the Lung.—Richardson reports two cases of this rare complication, in one of which it subsequently became necessary to open and drain a lung abscess. He also describes a case reported by La Play, where the resulting abscess was probably due to an embolism.

Sepsis.—It is probable that every case of tonsillectomy is complicated by some local infection, which is a natural consequence of laying bare two large granulating surfaces with some trauma in the presence of numerous bacteria. Certainly the three cases of cellulitis, one with the further issue of facial paralysis of five weeks' duration, reported by J. A. Stucky, would suggest this contingency. I have had five cases where the swelling of the glands in the parotid region was suffi-

ciently marked as to require attention, though none went to suppuration. The presence of oedema, which I have observed in some degree in at least 50 per cent of my cases, and have heard colleagues mention as a frequent complication in their work, however, is due to infection only in part, I believe, and has never caused any serious trouble in the convalescence. The removal of the tonsil *in toto* is likewise the removal of the support of the pillars and velum; hence, the relaxation and venous stasis contribute to the odema if they do not actually cause it. The flabby, immobile palate allows food and liquids to pass into the posterior nares, stimulating paresis; in fact, Sewell reports two cases of paresis of the soft palate, which suggest to me merely a severely relaxed, oedematous palate and a patient naturally unwilling to incur great distress by voluntarily putting on the stretch such sensitive parts. Mild general sepsis is fairly common, accompanied by a temperature of 100 F., but severe cases of systemic infection, involving gangrene of cervical muscles, or septic phlebitis with optic neuritis and delirium such as reported in Richardson's paper, as seen by L. W. Dean, are fortunately rare. Thus, Ballenger describes but two cases of his own, both due to the streptococcus; L. C. Deane says about 2 per cent of his cases showed this effect; Bauchacourt, in Dean's paper, one case of oedema of the glottis with death, and Martin two deaths, while Dean gives one death from septicaemia in his own practice. This occurred on the sixth day.

Emphysema.—Richardson's case, in which the air entered the subcutaneous area within an hour and cleared up in forty-eight hours, and the case of B. D. Parrish, where the emphysematous infiltration began a few minutes after the operation, and extended from the right eyelid down the cheek, face and chest to the last rib, are the only cases I find recorded. I am indebted to Dr. Richardson's paper for these, as it is.

Pneumonia and Pleurisy.—Scattered through the literature there is to be found an occasional report of pneumonia following a tonsillectomy, but as it is not always clear to the reader whether the death was due to the ether or to inspired matter it is not possible to classify them. However, Richardson re-

ports one pneumonia which proved fatal, and two pleurisies, neither of which was fatal. According to Crockett, there have been in recent years but two ether pneumonias at the Massachusetts Charitable Eye and Ear Infirmary, one of which died, but he has collected reports that show in the Boston neighborhood one death which he says was due to strangulation from vomitus, and one death he calls an anaesthetic death, though suspecting an enlarged thymus. A death from such a cause can in no way be attributed to the operation of tonsillectomy *per se*, but it is reported in this connection by the following, each of whom had one fatality: Dench, T. J. Harris, F. R. Packard. I have had one case of pneumonia which was certainly due to the manner of administration of the ether. His convalescence was very slow, and his temperature lasted for three weeks, but he suffered no permanent detriment.

Rheumatism.—Three days after removing the tonsils of a young boy a slight swelling was observed on the outer aspect of each ankle, and a temperature of 100 F. This subsequently rose to 101.2 F., and the case ran a typical course and history of acute articular rheumatism, with involvement of the knees as well as the ankles, lasting three weeks. I afterwards learned that he had been subject to such attacks every Winter and in cold weather at other seasons. Apparently the operative interference had released latent infection in the tonsillar crypts and adjoining lymphatics with metastatic evidences in the joints mentioned.

Nervous Phenomena.—Pfingst reports a case of hemiplegia which did not disappear till four months after operation, also one in which there were repeated attacks of hysterical strangulation and spasmodic flexing of the thighs (Bergh). Several months ago I saw, in consultation, an infant, fifteen months of age, from whose epipharynx lymphoid tissue had been curetted under chloroform anaesthesia, for the relief of mouth-breathing, the mother said. As an undoubted result of this unnecessary surgery, every afternoon since the operation this child had had violent attacks of spasmodic laryngitis, with complete arrest of respiration for as long as fifteen and even twenty seconds at a time, and all the cyanotic and cardiac dis-

turbance which usually accompanies such seizures. The child had a negative history, had never been ill and had parents of no taint whatever. Evidently the violence done her nervous equilibrium was great and causative of the attacks.

The rather severe edema of the uvula and soft palate following an enucleation of the tonsils in a neurotic woman twenty-five years of age had begun to disappear when I was sent for in great haste on the fifth day, as she was unable to speak even in a whisper. The cords appeared normal and moved freely on attempted phonation, and no cause other than hysteria seemed to me likely at the bottom of the aphonia. I tried to betray her into speaking by catching her off her guard, but knowing she was very desirous to go to Europe in a few days, I told her mother in her presence that her voice would return unimpaired by the second day at sea. This was sufficient to secure a cure, as promptly the second day, on getting up, her voice was found to be full and normal.

Injury to Pillars and Adnexa; Contractures; Functional Disturbances.—Laceration of the pillars is a frequent occurrence where they are friable from disease, or where much force, or rather misdirected force, is applied, and will materially increase hemorrhage, especially when the posterior pillar is the part damaged. Generally these wounds heal readily with no ill effects other than prolonged convalescence, but I had the misfortune several years ago to buttonhole an anterior pillar with the punch, and the wound did not close. Moreover, the cicatricial contractures were extensive, and the two pillars adhered together on each side, limiting the mobility of the velum in such a way as to render the patient's voice nasal from time to time, though not continuously. Since abandoning the use of the punch some three years ago I have had no such perforation or contractures. While I do not seek to escape the blame for my poor technique that brought about such a result, I find myself in accord with Sewell, who says that many of these bad cosmetic and functional results are due to the faulty administration of the anaesthetic. It is essential for good results in throat surgery that the patient should be kept quiet, the mouth free from blood and mucus and the anaesthetic apparatus out

of the operator's way. My case certainly bears out his theory, as the three conditions desired were conspicuously lacking.

I was rather surprised to read that Pfingst found it not unusual to buttonhole the pillars in the first twenty-five or thirty cases he had to report, and the uvula was at times removed along with the tonsils. This I have done once with the snare, and I have seen two eminent laryngologists do the same thing in this city. If the snare loop is made so small that the loosened tonsil can just be pulled through with slight force, it will be impossible to commit this surgical error, which I have likewise done once with the punch, as the parts were obscured with blood and the patient not well anaesthetized. In all I have had four cases in which the scar formation was marked, all due to indiscreet use of the tonsil punch. Sewell reports one case of adhesion of the velum to the pharyngeal wall, shutting off one naris from the oral cavity. I had a similar case in which the velum was adherent to the pharynx on both sides, leaving an opening in the mid-line which scarcely admitted my little finger. As he was a mental defective, an accurate history was not obtainable, other than that it was the result of a throat operation.

Where a tonsil is badly degenerated and the pillars share in the process, though the tonsil be removed with the utmost care and circumspection the rapid formation of granulations requires removal for weeks following convalescence, if deformities are to be prevented. Such has been my experience with tonsils the seat of infection spreading into peritonsilar abscess, and requiring removal as a prevention of recurrent attacks, though I try to prevent this new growth by local applications to the healing surface. One case, that of a woman of 45 years of age, required free punching out of large organized granulations three times in the course of three months.

The examination of a young man's throat for the relief of pain and offensive accumulations revealed the absence of a uvula and the presence of tonsil remnants on each side, though he had had a general practitioner remove the tonsils once, and again a specialist had tried to do it with the result already stated. Moreover the free edges were the seat of chronic inflammation, their surfaces ulcerated and covered with a pink-

ish and yellow material, and a glistening, tense white band of fibrous tissue bounded the free edge of the pillars on one side, up and across the palate and down the edge of the opposite pillars, holding the velum in a tight grip. As the patient was pale, weak and dispirited I would give no prognosis till I had a report from the pathologist, who examined a section from each tonsil, with a negative result. As there was no glandular involvement or temperature the tonsil base on each side was enucleated, revealing a sclerotic mass the size of an English walnut, true tonsil tissue. His voice is occasionally nasal, though the contracture is better than before my operation.

A clean removal of diseased or enlarged tonsils will at times increase the register of the singer's voice as much as two tones, but on the other hand it is unwise in the extreme to operate on the throat of a singer without first warning the patient what may be the outcome as to limitation of tone production. However carefully the removal be done, there will sometimes be a restraint placed on the palatal musculature by scar formation and adhesion of parts formerly free, such as to impair tone production and resonance. Such cases are on record; in fact, Stucky refers to one in his report.

Hudson Makuen goes into this particular point thoroughly and vigorously, laying stress on the carelessness with which the soft parts are handled. His explanation of why adhesions and fibrous formations alter the voice is particularly lucid, and I speak only in corroboration, since nothing can be added to his work save further evidence.

Whether we regard the enucleation of tonsils as an operation free from peril, or distinguished by an occasional mishap, at least the conviction forces itself upon us that it should not be entrusted to any but those experienced in its performance, and that the convalescence should be zealously followed. Moreover, it is essentially a hospital operation, to be done in a hospital or under the same rigid condition of asepsis and trained nursing.

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Candidate's Thesis.

CASE OF PURPURA HEMORRHAGICA.

BY MAX A. GOLDSTEIN, M.D.

Dyscrasias of the blood showing intense local manifestations in the mucous membrane of the mouth and buccal cavities are so infrequent and are so meagerly described in text-books and monograph-literature that I take this occasion to report minutely the following case of purpura hemorrhagica with local manifestations especially in the mouth:

M. M. F., aged 36 years, a Russian tailor, was admitted to the Medical Division of the Jewish Hospital of Saint Louis, December 1, 1911, and was referred to my department, December 3.

Present Condition: The patient was admitted bleeding from the left nostril, from the mouth and from the gums of the left superior maxilla. This bleeding began two days previous to the admission to the hospital, and the patient sought relief from a physician who packed the nose tightly with gauze. The bleeding in the nose continued through the dressing and also from the gums of the left upper jaw. Patient consulted a dentist who incised the mucous membrane of the gum and lip near the site of bleeding.

The bleeding seemed uncontrollable and the patient was admitted to the "in-patient" department of the Jewish Hospital and put to bed.

There was no marked pain except on manipulation about the nose, antrum, lips and gums; occasional headaches; no pains in the chest or abdomen; slight pain in the left thigh; appetite not impaired, but had not eaten for two days on account of bleeding in the mouth; sleep disturbed because of hemorrhage; bowel, constipated, passage black and tarry; urine, very dark red.

Previous History: No recollection of childhood dis-

eases; no serious illness up to the present time. Had tooth extracted last May, followed by marked bleeding. Since that time he noticed that he bled unusually freely from the slightest cut. No history of bleeding in any member of the family. Denies venereal diseases.

Plate I. (Before).

Examination: Middle aged man; small stature; expression, dull and anxious; color very sallow. Reddish and reddish-brown spots (pin-point to size of split pea) in skin on face

and generally scattered over body. These spots appeared suddenly, enlarged, and turned brown after a few days. *Eyes:* Color of sclera of eyes, yellowish, somewhat injected; pupils equal and react to light and accommodation. *Nose:* Left nostril markedly dilated and mucosa engorged and inflamed. Constant oozing through packing; area of bleeding not localized. No nasal symptoms prior to present attack. *Mouth:* Lips dry, swollen, dark-red spots on inner margin; similar spots on internal and external margin of gums, on left side of the upper and lower jaws, also on the surface of soft and hard palate, sublingual plica, and on uvula and fauces. Upper side, left jaw, shows very bad tooth. Bleeding under gum of first and second molars. Buccal cavity on left side very much swollen, pigmented and bloody. *Pharynx:* Difficult of inspection, as blood from posterior naris constantly trickled over surface. *Ears:* Negative. *Neck and Chest:* Negative except for purpuric spots. Heart-sounds good, no murmurs. *Abdomen:* No rigidity nor tenderness. *Liver* extended from seventh rib to slightly below the costal margin. *Spleen:* Not palpable. *Extremities:* No glandular enlargement, slight pain in left thigh; patella and plantar reflexes positive; Rombergism negative. Arteries soft, tension good.

Clinical Record, December 3, 1911: Numerous purpuric spots appearing on flexor surface of arms and legs, and on face. Nose and gums packed four to five times in twenty-four hours with gauze strips soaked in adrenalin chlorid. Tincture opii internally M X. Two tubes of antidiphtheritic serum injected subcutaneously. Calcium lactate (gr. 10) every three hours; gelatin by mouth; liquid nourishment only; ice caps and cold compresses constantly applied to head and face. Temperature 98 degrees-98.6 degrees (axillary).

December 4: Nose and gums packed with 25 per cent ferrous subsulphate. Parts cleaned with hydrogen peroxide.

December 5: Nose packed with bismuth subnitrate gauze and dusted with aristol. Patient felt slightly better and less alarmed.

December 6: All packs removed and parts dusted with aristol. Still slight oozing. Left eyelids and infra-orbital

region infiltrated reddish-yellow; similar blotches on exterior surface of left arm. Temperature 98 degrees-99.8 degrees (axillary). Calcium lactate, internally administered, continued in 10 grain doses every three hours. Olive oil t. i. d.

Plate II. (After).

December 8: Urine of normal color the first time since admission of patient to hospital. Temperature 98.6 degrees-100.2 degrees. Sero-sanguinous discharge from left nostril.

Left side of nose, superior maxillary process, eyelids, and bridge of nose much swollen and bluish in color. Very little evidence of bleeding. Petechial spots still present on palate and gums.

December 10: Swelling over superior maxilla decreased in upper but increased in lower areas. On gingival mucous membrane, just above first molar tooth on left side, fluctuating tumor, the size of a large hazel-nut; pain on slight pressure. Tumor incised, slight amount of purulent matter expressed. Tumor area painted with tincture of iodin. Temperature 99.2 degrees.

December 13: General condition of patient much better; swelling almost disappeared from face; only traces of spots on gums and palate remain. Purpuric spots in skin of body have disappeared.

December 16: Temperature normal for three days. Patient much improved; discharged from "in-patient" department.

Blood Examination, December 3: Hemoglobin 75 per cent; leucocytes 15,000.

December 4: Red count 5,750,000; hemoglobin 80 per cent.

December 9: Red count 4,500,000; white count 14,600; hemoglobin 85 per cent.

Urine: Examined daily; showed neither albumen nor sugar, nor increase of indican.

Fecal Examination: Stools very dark, hard, tar-like. Guiac test very positive during bleeding; cleared rather abruptly.

Radiograms: First X-ray was taken December 7, 1911; the second X-ray was taken December 15, 1911.

This case, seen in consultation with Drs. Tuholske, Friedman, Jonas, Lippmann, Meyers and Deutsch, was diagnosed as a rather unusual form of purpura hemorrhagica of unknown etiology. The clinical evidences were sufficiently marked to warrant this diagnosis.

The factors of unusual interest from a laryngological point of view were the localization of the unusual and sharply marked purpuric blotches in the mouth and buccal cavity, the spontaneous bleeding from the nose and gums as possible

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CASE OF DR MAX GOLDSTEIN

etiological factors, the apparent ineffectiveness of internal therapy until the injection of serum had been made, and a further possible etiological factor in the left nasal fossa and maxillary antrum as shown by the radiograms.

The colored plate, drawn at the bed-side, gives a fairly accurate idea of the areas involved in the localized purpuric infiltration.

It is a significant feature of this case that all of the areas of pigmentation, whether in the mucosa or skin of body and extremities, appear on the left side.

In the mouth and buccal cavity this intense pigmentation is outlined by a sharp straight line to the vertical edge of the median raphe in the hard and soft palate and the frenum in the gums of the lower jaw. The deepest coloration of bluish red appears over the hard palate corresponding to the area over the floor of the nose; this might, perhaps, be associated with the original pathology in the alar wall of the nose and nasal wall and floor of antrum.

Repeated inspection of the nose of the patient after convalescence showed the septal mucosa on the left side perfectly normal, but the left alar nasal surfaces about the middle turbinal body and inferior meatus showed similar irregular areas of erosion.

The left maxillary antrum was washed out several months after the discharge of the patient, and the wash came away perfectly clear. These observations are also substantiated by the second radiogram as compared with the first, in which obscurity and possible fluid contents (perhaps blood) were evident.

The cause of the spontaneous bleeding from the nose was not ascertained. When the patient was at first seen at the hospital there was an ulceration or erosion of the gums about the first molar tooth, which was painful to the slightest manipulation, and bled freely.

Of all the therapeutic measures carried out in the conduction of this case we have reason to believe that the only effective agent utilized was the injection of two tubes (10 cem. each) of anti-diptheritic serum. Thirty-six hours after injection of the

serum all packings removed from the nose and very little oozing followed.

A critical examination and comparison of the two radiograms, the first taken during the hemorrhagic activities in the nasal and oral cavities, and the second after the patient was discharged from the hospital showed decided differences in appearance. In the first radiogram there is a distinct area of shadow in the left nasal fossa and left antrum, and there is also some evidence of abnormality indicated in the shadow of floor of left nose, antrum and appearance of bone of left superior maxilla; in the second radiogram both nares appear more nearly of the same shading, as do the floor and nasal wall of antrum and area over the left maxilla.

The case is unique in kind, as I have searched the literature of purpura hemorrhagica carefully and have failed to find any mention of localization in the area of the nasal and oral cavities.

I am indebted to Dr. Joseph O'Reilly for the careful bedside notes furnished in this case.

Candidate's Thesis.

THE REQUIREMENTS OF AN IDEAL OPERATION FOR CHRONIC FRONTAL SINUSITIS.

BY O. A. M. McKIMMIE, M.D.

The earlier radical operations on the frontal sinus had as an object the complete obliteration of the cavity.

Many modifications of the Killian operation have in view the preservation of this air space and actual extension of its limits by removal of the ethmoid cells in part or as a whole, and if necessary the removal of the anterior wall of the sphenoid. The operations proposed by Knapp and Bryant are of this type.

In this short discussion no further mention will be made of operations by the intra-nasal route, as I do not consider that they fulfill the ideal requirements of a procedure intended to cure and not merely to ameliorate a suppurative sinusitis.

The ideals to be sought are:

1. Efficient and permanent drainage;
2. An operative wound of such character as to give free access to every part of the diseased area;
3. Removal of contiguous air cells—usually diseased—so as to markedly enlarge the communication between the frontal and the nose;
4. Preservation of all tissues whose removal is not necessary to eradicate disease or to permit perfect drainage;
5. A technique permitting removal of all tissues under direct inspection or guided by the finger in contact with the tissues being removed; or a combination of both inspection and palpation;
6. The avoidance of external deformity.

The operation to be described I believe fulfills the requirements stated.

When possible a skiagram of the frontal region should be made and the vertical and lateral extent of the sinus made clear.

When such an aid is not available, the first bone removal should be made below the orbital ridge, thus permitting the outlining of the cavity in vertical and horizontal direction by means of the probe.

The skin incision is made in the mid-line of the unshaved eyebrow and extends from its temporal limit to its nasal end and then turns sharply downward, about a quarter of an inch from the ridge of the nose, until it reaches a point an eighth of an inch below the lowest limit of orbit.

No marks or superficial cross incisions are made for the purpose of aiding subsequent adaptation of the skin margins; they have always impressed me as being unnecessary and as adding a possible danger of deformity by keloid degeneration.

From a point slightly external to the supraorbital notch the periosteum is incised following the skin incision to its lower limit, and the skin and periosteum elevated, exposing the external portion of the nasal bone, the frontal process of the superior maxilla and the lachrimal bone. The elevation outward must be extended to such an extent as to give free working space even if the pulley of the superior oblique has to be detached in so doing.

If one is working with the aid of a skiagram the next step is the elevation of the skin fom the frontal wall of the sinus to such an extent as to permit the outlining of the bone bridge by incising the periosteum from the outer third of the skin incision to within an eighth of an inch of the middle line of the forehead.

If no skiagram has been made it is much safer to first remove the bone exposed below the supra-orbital ridge with a straight edged chisel and then having ascertained the extent of the sinus to proceed with the uncovering of the frontal wall.

A bridge of bone, with its periosteum attached, at least one-half inch wide should be left, if study of a skiagram or determination of the extent of the cavity make it seem that such a

width will not interfere with thorough inspection of every part of the sinus, or if the vertical dimenson of the cavity permits.

The prevention of deformity is dependent on preservation of a broad bone bridge and the overhang of the upper margin of the window made by the removal of part of the anterior wall.

When the upper edge of the bridge has been outlined with the V-shaped chisel, the periosteum above it, together with the skin, should be elevated together over the whole area of the sinus.

With chisel and rongeur so much of the frontal wall should be taken away as to give access to every recess of the cavity, bony septa should be removed and all granulation masses and necrotic areas taken away, due attention being given to the inner surface of the bone bridge.

Especial attention should be given to the small cells frequently found in the upper portion of the inner wall of the cavity, but for the purpose of avoiding even slight insinking of the skin the overhang of bone, left when the window in the front wall is made, should be preserved if possible.

The next step is the removal of the ethmoid cells or so much of them as seems necessary. This is best started with a curette passed from above the bridge and used with a boring motion to enlarge the infundibulum. The little finger of the other hand may be passed into the nostril so as to serve as a guide and to prevent injury to the septum.

If a preliminary removal of the middle turbinate has not already been done it should now be removed by whatever method the operator prefers and the ethmoid cells taken away. If, as in most cases, they are diseased, the whole anterior and posterior series should be ablated, leaving the paper plate, and if possible, the bone and mucous membrane of the superior turbinate.

If it seems necessary, the sphenoid may now be easily opened and its anterior wall removed.

The ethmoid cells are most easily attacked by a punch forceps passed through the opening under the bridge and guided by a finger in the nose or the finger may be passed through the external opening and the punch through the nostril.

After the free bleeding has ceased the frontal cavity should

be packed with a long strip of gauze whose end is brought out of the nostril and marked in some way for identification. After this the rest of the nasal chamber should be packed with gauze. The next step is closure of the external wound and the application of a fluff dressing and bandage.

After twenty-four hours the lower nasal dressing is removed and not replaced unless required to stop hemorrhage. The gauze strip in the frontal cavity is then pulled down until about half is removed and cut off, the remainder being removed on the second day. The skin sutures are removed on the fifth day. The after treatment consists of such cleansing measures as are necessary to prevent the formation of crusts, the removal of such as may form and further the caring for such granulation masses as may block drainage.

The older methods of operation on the frontal sinus caused impaired nasal resonance by obliteration of the cavity; this method also causes temporarily a change in the voice tones by increasing the resonance spaces on one side of the head.

Nothing new is claimed for the operation described. It is really a combination of what has seemed best in the work of several operators so as to fulfill most nearly the ideals stated earlier in this paper.

So far as the writer knows the idea of saving the upper inner portion of the ethmoid body—the superior turbinate and its mucous membrane—is original. The purpose is two-fold; first to provide a fairly large and well nourished epithelial edge from which epithelialization of the cavity may spread, and secondly to interpose a broader barrier to the possible spread of infection to the anterior fossa of the cranial cavity by way of the cribriform plate.

Epithelial covering of the enlarged frontal cavity is hastened if such fragments of its lining membrane as seem healthy or are at least not granular are not removed.

The following case report and accompanying photograph illustrate the results which this operation give:

Miss A. M., adult, was seen in consultation on the afternoon of June 1, 1912. She presented a fistula near the inner margin of the right orbit from which thin pus was coming and the

use of a lachrymal syringe through the fistula brought away not only fluid pus but large ropy masses of muco-pus.

There was marked swelling of the orbital tissues and proptosis. Examination of the nose revealed a very narrow nasal chamber but no purulent discharge, and a diagnosis of chronic closed empyema of the frontal and ethmoid, with external rupture, was made. The patient's condition seemed critical and I operated on her the same night.

At the time of the operation on her sinuses her condition was such that no skiagram was made. The size of the frontal, however, could be easily determined with a bent probe passed through the fistula. The operation was conducted after the method described and was without incident except the finding of an extremely large ethmoidal bulla which projected upward like a dome and contained about a drachm and a half of very thick pus. It was necessary to remove a very large part of her

frontal wall, but a bridge more than half an inch wide was saved and the overhang of the upper margin of the window preserved.

The previous history of this patient is very interesting.

Although I did not at first recognize her, she subsequently told me that when a little girl she had been brought to see me by her family physician on account of some nasal trouble. About two years before I operated on her she had had the lachrymal sack removed for a chronic purulent dacryocystitis by the surgeon who put her in my care for her sinus trouble.

At this operation he found what was apparently a long extension of the sack upward.

His operation wound healed and remained in perfect condition until a short time before she came under my care.

I found it necessary to remove the lachrymal bone and part of the paper plate anteriorly.

The ethmoid cells were completely removed but the superior turbinate saved. The sphenoid was also opened so that when the operation was completed the nasal chamber was one large cavity.

The after history was uneventful. The orbital swelling and proptosis subsided gradually and the general condition of the patient improved rapidly.

The accompanying photograph was taken March 9th, 1913, by Dr. Frank A Smart, Resident Physician at the Episcopal Eye, Ear and Throat Hospital. It is printed from an absolutely un-retouched plate. The eyebrow was parted at the time the picture was made, with the intention of showing the scar at its worst.

Remarks on Technique.

To prevent blood from flowing into the nasopharynx during the work within the sinus proper the nose should be packed with as many superimposed cotton rolls soaked in adrenalin solution as are necessary to fill the nasal chamber. The lowest roll should be long enough to extend to the posterial opening and the high ones shorter. As they are to be left in place until

the removal of the ethmoid cells is commenced, they should be squeezed free of any excess of adrenalin, so that there may be no unnecessary systemic action by swallowing solution which might pass into the throat.

Special care should be taken to avoid invading the orbit by fracture of any portion of the paper plate of the ethmoid. In removing the ethmoid cells it is very easy to fracture this thin plate of bone and set up an acute infection in the eye socket. On the other hand, if this cavity is already involved, so much of the bone as is diseased, or as it may be necessary to remove to provide for free drainage, should be taken away.

In Memoriam

These pages are respectfully dedicated to the memory of those whom Providence has seen fit to remove from our midst during the year. Expressions of sorrow inadequately define our loss, and a recital of their achievements is but a repetition of the history of Laryngology in America. Their work is common property and known to all.

CHARLES HUNTOON KNIGHT

Born November 22, 1849, in Easthampton, Mass. Graduated from Williams College with the Class of 1871. Received the degree of M.D. from the College of Physicians and Surgeons, New York, in 1874. He became a Fellow in the American Laryngological Association in 1885. He was elected Secretary in 1889, in which capacity he served until 1896, when he was elected President. Married to Miss Lucy Tolford Mackenzie, of New York, on June 28, 1893, who survives him.

On April 29, 1913, Dr. Knight died at his residence, 55 East Ninety-third street, New York, after a prolonged illness of many years, endured with uncomplaining fortitude.

ERNEST L. SHURLY

Born June 11, 1846, in Buffalo, N. Y. Was graduated with degree of M.D. from the University of Buffalo in 1866. He became a Fellow in the American Laryngological Association in 1878 and was one of its founders. Elected President of the Association in 1884. He was married to Miss Elizabeth Pultz April 13, 1868. Dr. Shurly died at his home in Detroit, May 10, 1913.

ARTHUR AMES BLISS

Born in Northampton, Mass., July 13, 1859. Received the degree of A.M. from Princeton College in 1880, and the degree of M.D. from the University of Pennsylvania in 1883. He was married to Miss Laura Newhaus, of Vienna, Austria, October, 1893. He became a Fellow in the American Laryngological Association in 1893. Dr. Bliss died May 1, 1913.

C. E. BEAN

It is with regret that no data has been obtainable concerning Dr. Bean. A note is on file in Dr. Newcomb's handwriting, stating that Dr. Bean's application for membership had been lost. He became a Fellow of this Association in 1880, Vice President in 1889, and died in the year 1913.

SECRETARY'S REPORT.

The secretary begs leave to report that during the past year the association has lost by death Doctors C. E. Bean, of St. Paul; James E. Newcomb and Charles H. Knight, of New York, and Arthur Bliss, of Philadelphia. Data relative to Dr. Bean has been unobtainable, though efforts have been made to secure the same. A committee consisting of several fellows of the association attended the funeral of Dr. Newcomb in New London, Conn., and the secretary sent a floral tribute in the name of the association, which was acknowledged by Mrs. Newcomb in the following letter:

"Will you kindly extend to the fellows of the American Laryngological Association the thanks of Dr. Newcomb's entire family for the beautiful wreath which you sent us last Thursday? Knowing how near to my dear husband's heart this organization has always been, I wish to extend to its fellows my deepest appreciation of their last token of love to him and sympathy to me in my great bereavement."

Dr. Charles H. Knight, our retired fellow, died on April 29th. He was elected a fellow of this association in 1885 and served as secretary from 1889 to 1895. He was president in 1896. He was made a retired fellow in 1912. His interest in the society had always been active, and it was even with reluctance that he accepted the honorary title of retired fellow. A floral tribute was sent in the name of the association and notification of the funeral services to the New York and Brooklyn fellows. Eight of these gentlemen responded to the call and met to pay the last tribute to our departed fellow.

These four deaths, together with the three fellows who have become retired fellows, leave us a total active fellowship of eighty-seven. There have been no resignations nor any deaths amongst our honorary and corresponding fellows. We have now four honorary and twenty-four corresponding fellows.

The secretary again calls the attention of the members to the importance of returning their corrected discussion as early as is possible, as the publishers of the Transactions extend a Summer rate to the society, and when their work is carried beyond September an extra charge is made, which expense can be obviated by the fellows themselves.

Notification of the celebration of the sixtieth birthday of Professor Chiari, our honorary fellow, was sent to the president by Dr. Marschik, but it was received too late for a contribution to be made in time for the affair. An acknowledgment, however, of the receipt of the invitation was sent to Dr. Marschik, wishing all prosperity and happiness to our esteemed and honored fellow, in the name of the association. Professor Chiari answered the expressions of congratulation in the following words:

"Through Dr. Marschik I received the kind wishes of the members of the renowned American Laryngological Association, whose honorary fellow I call myself with pride. I beg you to transfer my best thanks to the esteemed members, and, believe me, very sincerely yours, OTTO CHIARI."

Report of council meeting, January 11, 1913: The place and time of the annual meeting of the association was determined, and the gentlemen subsequently elected to membership were recommended to the association. For corresponding fellows, Doctors Turner, Von Eiken and Denker, were submitted to the association for election.

Report of second council meeting, May 4, 1913, held at Washington, D. C., Dr. G. A. Leland in the chair: It was moved and carried that it be recommended to the association that a redraft of the articles of incorporation be made to conform with the New York State law. It was also moved that it be recommended to the society that the annual dues be increased to meet the increasing expenses of the secretary and treasurer's office, and also of the publication of the Transactions. Other matters of discussion appear in the minutes of the secretary.

EXECUTIVE SESSION.

Auditing Committee—Drs. Hopkins and Packard. Nominating Committee—Drs. Ingals, Swain, Roe, Jackson and Shurly.

It was moved and carried that the secretary cast one ballot for the election of the officers as nominated.

De Roaldes Prize Essay Committee—Drs. Makuen, Logan and Kyle. The committee reported that neither essay was of sufficient merit to warrant the presentation of the award.

Election of New Members. Active Fellows—Drs. Ralph Butler, Rockwell Coffin, Virginius Dabney, Max Goldstein, Hanau Loeb, Oscar McKimmie and Dunbar Roy. Corresponding Fellows—Drs. Logan Turner, of Edinburgh; Carl von Eiken, of Geissen, and Alfred Denker, of Halle. Retired Fellow—Dr. A. W. De Roaldes.

By a unanimous vote these fellows were elected to membership in the society.

There were registered at the session fifty-eight fellows.

Drs. Coakley and Smith were appointed a committee of two to secure proper legal advice and to reconstruct the articles of incorporation and by-laws to conform with the New York State requirements.

The Publication Committee, consisting of Drs. Shambaugh, Coolidge and Kyle, were retained in the capacity and requested to still further investigate the advisability of founding a new journal of laryngology.

The Tonsil Investigation Committee, which consisted of Drs. Richards, Clark and Swain, had added to it Drs. Ingals and Wright. It was moved and carried that the matter of investigation be submitted to the Sprague Memorial Institute of Chicago and that the expense of the committee come out of the general funds of the association.

The Ozena Investigation was dropped, and it was voted by the society that one-third of the expense incurred by the chairman of the committee be defrayed by the society.

International Medical Congress—Dr. Thomas Hubbard, the incoming president, was appointed as the representative of the association at this congress, and it was decided that all fellows wishing to be delegates to the congress should apply to the president or secretary for a written form engraved for the purpose.

A committee consisting of Drs. Wood and Wilson was appointed to investigate cancer of the upper respiratory tract.

Since the May meeting our esteemed and honored fellow, Dr. E. L. Shurly, has been added to the great loss we have sustained in the last two years. His death took place Saturday, May 17, 1913.

Librarian's Report.

Your librarian has the honor to report that since the last meeting five bound and seven unbound books and ninety-five pamphlets have been received and deposited in the library of the Surgeon General of the United States Army.

TREASURER'S ANNUAL STATEMENT.

RECEIPTS.

Balance from old account	$378.82
Dues collected	810.30
Sale of transactions	36.05
Total	$1,225.17

EXPENDITURES.

Lantern exhibition	$5.00
Stenographer, 1912	100.00
Dies for DeRoaldes Medal	175.00
Express	9.00
Stenographer	9.30
Stationery (Secretary)	21.49
Slips and envelopes (Secretary)	4.50
Packing and shipping books	5.00
Flowers (Dr. Newcomb's funeral)	20.00
Paper and printing (Treasurer)	3.25
Stenographer (Treasurer)	3.20
Transactions and mailing	747.59
Stenographer (Secretary)	9.09
Programs and distribution	40.55
Collecting checks	1.50
Translating Natier's paper	5.00
Postage (Treasurer)	2.68
Dr. C. H. Knight's dues returned	10.00
Total expenditures	$1,172.15
By balance in bank	53.02
Total	$1,225.17

ASSETS OF ASSOCIATION.

INVESTED.

DeRoaldes Fund	$500.00	
Accrued interest	112.49	
		$612.49

CURRENT.

Unpaid dues	$110.00	
Bank balance	53.02	
		$163.02
Total		$775.51

LIABILITIES—NONE.

Respectfully submitted,

J. PAYSON CLARK, Treasurer.

Washington, May 5, 1913.

Audited and approved.

F. E. HOPKINS.
FRANCIS R. PACKARD,
Auditing Committee.

LIST OF ACTIVE FELLOWS.

1914.

1906. Ballenger, William L., Columbus Memorial Building, Chicago, Ill.
1908. Barnhill, John F., 328 Delaware St., Indianapolis, Ind.
1904. Berens, T. Passmore, 35 Park Ave., New York.
1893. Birkett, Herbert S., 252 Mountain St., Montreal.
1901. Brown, J. Price, 28 College St., Toronto Can.
1892. Brown, Moreau R., 34 Washington St. Chicago, Ill.
1891. Bryan, Joseph H., 818 17th St., Washington, D. C.
1913. Butler, Ralph, 1824 Chestnnut St., Philadelphia, Pa.
1889. Casselberry, William E., 15 E. Washington St., Chicago, Ill.
1896. Chappell, Walter F., 7 E. 55th St., New York.
1897. Clark, J. Payson, 71 Marlborough St., Boston, Mass.
1902. Coakley, Cornelius G., 53 West 56th St., New York.
1899. Cobb, Frederic C., 11 Marlborough St., Boston, Mass.
1905. Coffin, Lewis A., 156 West 58th St., New York.
1913. Coffin, Rockwell A., University Club, Boston, Mass.
1878. Cohen, J. Solis, 1824 Chestnut St., Philadelphia, Pa.
1893. Coolidge, Algernon, Jr., 613 Beacon St., Boston, Mass.
1913. Dabney, Virginius, 1633 Connecticut Ave., Washington, D. C.
1882. De Blois, Thomas Amory, 48 Gloucester St., Boston, Mass.
1881. Delavan, D. Bryson, 40 East 41st St., New York.
1892. Farlow, John W., 234 Clarendon St., Boston, Mass.
1901. Freeman, Walter J., 1832 Spruce St., Philadelphia, Pa.
1905. Freer, Otto T., 34 Washington St., Chicago, Ill.
1879. French, Thomas R., 150 Joralemon St., Brooklyn, N. Y.
1897. Frothingham, Richard, 616 Madison Ave., New York.
1909. Getchell, Albert C., 6 Linden St., Worcester, Mass.
1907. Gibb, Joseph S., 1907 Chestnut St., Philadelphia, Pa.

1880. Gleitsmann, Joseph W., 616 Madison Ave., New York.
1913. Goldstein, Max, 3858 Westminster Place, St. Louis, Mo.
1898. Goodale, Joseph L., 258 Beacon St., Boston, Mass.
1905. Grayson, Charles P., 251 South 16th St., Philadelphia, Pa.
1906. Greene, D. Crosby, Jr., 483 Beacon St., Boston, Mass.
1905. Halstead, Thomas H., 831 University Block, Syracuse, N. Y.
1896. Hardie, T. Melville, 34 Washington St., Chicago, Ill.
1903. Harris, Thomas J., 104 East 40th St., New York.
1878. Hartman, Jacob H., 5 West Franklin St., Baltimore, Md.
1888. Hinkel, F. Whitehill, 581 Delaware Ave., Buffalo, N. Y.
1907. Holmes, Christian R., 8 East 8th St., Cincinnati, Ohio.
1893. Hope, George B., 159 West 72d St., New York.
1895. Hopkins, Frederick E., 25 Harrison Ave., Springfield, Mass.
1895. Hubbard, Thomas, 515 Nicholas Building, Toledo, Ohio.
1878. Ingals, E. Fletcher, 15 E. Washington St., Chicago, Ill.
1904. Ingersoll, John M., 1021 Prospect Ave., Cleveland, Ohio.
1907. Jackson, Chevalier, 1018 Westinghouse Building, Pittsburgh, Pa.
1878. Johnston, Samuel, 204 West Monument St., Baltimore, Md.
1898. Kyle, D. Braden, 1517 Walnut St., Philadelphia, Pa.
1880. Langmaid, Samuel W., 143 Newbury St., Boston, Mass.
1894. Leland, George A., 354 Commonwealth Ave., Boston, Mass.
1913. Loeb, Hanau W., 537 North Grand Ave., St. Louis, Mo.
1897. Logan, James E., 1208 Wyandotte St., Kansas City, Mo.
1888. Lowman, John H., 1807 Prospect Ave., S. E. Cleveland, Ohio.
1911. Loekard, Lorenzo B., Metropolitan Bldg., Denver, Colo
1886. MacCoy, Alexander W., 216 South 15th St., Philadelphia, Pa.
1883. Mackenzie, John N., 605 Washington Place, Baltimore, Md.
1898. Maknen, G. Hudson, 1627 Walnut St., Philadelphia, Pa.
1896. Mayer, Emil, 40 East 41st St., New York.

1899. McKernon, James F., 62 West 52d St., New York.
1918. McKimmie, Oscar A. M., 1330 Massachusetts Ave., N. W., Washington, D. C.
1904. Mosher, Harris P., 828 Beacon St., Boston, Mass.
1901. Myles, Robert C., 46 West 38th St., New York.
1903. Packard, Francis R., 304 South 19th St., Philadelphia, Pa.
1906. Pierce, Norval H., 31 Washington St., Chicago, Ill.
1878. Porter, William, 3886 Washington Ave., St. Louis, Mo.
1908. Randall, B. Alexander, 1717 Locust St., Philadelphia, Pa.
1903. Renner, W. Scott, 341 Linwood Ave., Buffalo, N. Y.
1897. Rhodes, J. Edwin, 100 State St., Chicago, Ill.
1884. Rice, Clarence C., 123 East 19th St., New York.
1905. Richards, George L., 84 North Main St., Fall River, Mass.
1902. Richardson, Charles W., 1317 Connecticut Ave., Washington, D. C.
1879. Roe, John O., 44 Clinton Ave. South, Rochester, N. Y.
1913. Roy, Dunbar, Grand Opera House, Atlanta, Ga.
1907. Shambaugh, George E., 100 State St., Chicago, Ill.
1909. Shurly, Burt R., 32 Adams Ave. West, Detroit, Mich.
1909. Sluder, Greenfield, 3542 Washington Ave., St. Louis, Mo.
1892. Simpson, William K., 952 Lexington Ave., New York.
1908. Smith, Harmon, 44 West 49th St., New York.
1911. Stout, George C., 1611 Walnut St., Philadelphia, Pa.
1889. Swain, Henry L., 232 York St., New Haven, Conn.
1903. Theisen, Clement F., 172 Washington Ave., Albany, N. Y.
1892. Thrasher, Allen B., The Groton, 7th and Race Sts., Cincinnati, Ohio.
1892. Wagner, Henry L., 518 Sutter St., San Francisco, Cal.
1892. Watson, Arthur W., 126 South 18th St., Philadelphia, Pa.
1908. Wilson, J. Gordon, 104 South Michigan Ave., Chicago, Ill.
1906. Winslow, John R., The Latrobe, Charles and Reade Sts., Baltimore, Md.

1905. Wood, George B., 129 South 18th St., Philadelphia, Pa.
1890. Wright, Jonathan, N. Y. Post. Grad. Med. School, 20th St., and 2d Ave., New York.

Honorary Fellows.

1910. Chiari, Ottokar Von, Bellariastrasse 12, Vienna, Austria.
1910. Massei, F., 4 Piazza Municipio, Naples, Italy.
1910. Moure, E. J., 25 Cours du Jardin Public, Bordeaux, France.
1910. Semon, Sir Felix, Rignall, Great Missenden, Bucks, England.

Corresponding Fellows.

1880. Bruns, Paul Von, Tübingen, Wurtemberg, Germany.
1901. Collier, Mayo, 145 Marley St., Cavendish Square, London, W., England.
1890. De La Sota Y Lastra, R., 7 Calle de Torqueros, Seville, Spain.
1913. Denker, Alfred, Martinsberg 11, Halle, Germany.
1893. Desvernine, C. M., Cuba 52, Havana, Cuba.
1913. Eicken, Carl von, Friedrichstrasse 14, Giessen, Germany.
1901. Grant, Dundas, 18 Cavendish Square, London W., Eng.
1902. Heymann, Paul, 60 Lutzowstrasse, Berlin W., Germany.
1908. Killian, Gustav von, Lutzow Ufer 3, Berlin W. 10, Germany.
1892. Krause, H., San Remo, Italy.
1902. Lermoyez, Marcel, 20 Rue de la Boetie, Paris, France.
1897. Luc, H., 54 Rue de Varenne, Paris, France.
1903. McBride, P., 16 Chester St., Edinburgh, Scotland.
1896. MacDonald, Greville, 85 Harley St., Cavendish Square, London W., England.
1894. MacIntyre, John, 179 Bath St., Glasgow, Scotland.
1896. Mygind, Holger, 31 Havnegade, Copenhagen, Denmark.
1901. Natier, Marcel, 10 Rue de Ballachasse, Paris 7e, Paris, France.

1910. Navratil, Emerich Von, iv Rigyoteri, Buda-Pesth, Hungary.
1903. Onodi, A., o'Uctza 12, Buda-Pesth, Hungary.
1910. Rethi, Leopold, IX Garnisongasse 7, Vienna, Austria.
1894. Sajous, C. E., 2043 Walnut St., Philadelphia, Pa.
1896. Schmiegelow, Ernest, 18 Norregade, Copenhagen, Denmark.
1902. Thomson, Sir St. Clair, 64 Wimpole St., London W., England.
1903. Tilley, Herbert, 72 Harley St., Cavendish Square, London W., England.
1913. Turner, Logan, 27 Walker St., Edinburgh, Scotland.
1901. Wingrave, Wyatt, 11 Devonshire St., Portland Place, London W., England.
1894. Wolfenden, R. Norris, 76 Wimpole St., Cavendish Sq., London W., England.

Retired Fellows.

1878. Bosworth, Francke H., 41 Park Ave., New York.
1903. Lincoln, William, 210 Lennox Bldg., Cleveland, Ohio.
1893. Roaldes, Arthur W. De, 624 Gravier St., New Orleans, La.

PAST OFFICERS.

Presidents.

(Year of Election.)

1879. Louis Elsberg.
1880. J. Solis-Cohen.
1881. F. I. Knight.
1882. G. M. Lefferts.
1883. F. H. Bosworth.
1884. E. L. Shurly.
1885. Harrison Allen.
1886. E. Fletcher Ingals.
1887. R. P. Lincoln.
1888. E. C. Morgan.
1889. J. N. Mackenzie.
1890. W. C. Glasgow.
1891. S. W. Langmaid.
1892. M. J. Asch.
1893. D. B. Delavan.
1894. J. O. Roe.
1895. W. H. Daly.
1896. C. H. Knight.
1897. T. R. French.
1898. W. E. Casselberry.
1899. Samuel Johnston.
1900. H. L. Swain.
1901. J. W. Farlow.
1902. J. H. Bryan.
1903. J. H. Hartman.
1904. C. C. Rice.
1905. J. W. Gleitsmann.
1906. A. W. de Roaldes.
1907. H. S. Birkett.
1908. A. Coolidge, Jr.
1909. J. E. Logan.
1910. D. Braden Kyle.
1911. James E. Newcomb.
1912. George A. Leland.
1913. Thomas Hubbard.

Vice-Presidents.

1879. F. H. Davis.
1880. W. C. Glasgow, J. O. Roe.
1881. E. L. Shurly, W. Porter.
1882. C. Seiler, E. F. Ingals.
1883. S. W. Langmaid, S. Johnston.
1884. J. H. Hartman, W. H. Daly.
1885. H. A. Johnson, G. W. Major.
1886. E. C. Morgan, J. N. Mackenzie.
1887. J. N. Mackenzie, S. W. Langmaid.
1888. W. C. Glasgow, C. E. Sajous.

1889. E. Holden, C. E. Bean.
1890. J. O. Roe, J. H. Hartman.
1891. M. J. Asch, S. Johnston.
1892. S. Johnston, J. C. Mulhall.
1893. J. C. Mulhall, W. E. Casselberry.
1894. C. C. Rice, S. H. Chapman.
1895. J. Wright, A. W. de Roaldes.
1896. T. M. Murray, D. N. Rankin.
1897. A. W. MacCoy, H. S. Birkett.
1898. J. W. Farlow, F. W. Hinkel.
1899. T. A. DeBlois, M. R. Brown.
1900. H. L. Wagner, A. A. Bliss.
1901. J. W. Gleitsmann, D. Braden Kyle.
1902. G. A. Leland, T. Melville Hardie.
1903. J. H. Lowman, W. Peyre Porcher.
1904. Thomas Hubbard, W. J. Freeman.
1905. J. L. Goodale, C. W. Richardson.
1906. G. H. Makuen, A. B. Thrasher.
1907. J. P. Clark, J. E. Rhodes.
1908. E. Mayer, F. R. Packard.
1909. C. G. Coakley, H. P. Mosher.
1910. Robert C. Myles, J. M. Ingersoll.
1911. F. C. Cobb, B. R. Shurly.
1912. A. W. Watson, W. Scott Renner.
1913. F. E. Hopkins, George E. Shambaugh.

Secretaries and Treasurers.

1879. G. M. Lefferts.
1882. D. B. Delavan.
1889. C. H. Knight.
1895. H. L. Swain.
1900. J. E. Newcomb.
1911. Harmon Smith.

Treasurer.

1912. J. Payson Clark.

Librarians.

1879. F. H. Bosworth.
1883. T. R. French.
1903. J. H. Bryan.

PAST MEMBERS.

Honorary Fellows.

*1880. Senor Manuel Garcia, London, England.
*1883. Sir Morell Mackenzie, London, England.
*1907. Leopold von Schroetter, Vienna, Austria.
*1904. B. Fraenkel, Berlin, Germany.

Corresponding Fellows.

*1892. Lennox Browne, London, England.
*1887. A. Gouguenheim, Paris, France.
*1894. Edgar Holden, Newark, N. J.
*1881. Carlo Labus, Milan, Italy.
*1880. Wilhelm Meyer, Copenhagen, Denmark.
*1901. Moritz Schmidt, Frankfort-a-Main, Germany.
*1881. Pugin Thornton, London, England.
*1881. R. Voltolini, Breslau, Germany.
*1880. W. McNeil Whistler, London, England.
‡1881. Sir Felix Semon, Bucks Co., England.
‡1887. E. J. Moure, Bordeaux, France.
‡1892. F. Massei, Naples, Italy.
‡1896. Ottokar Chiari, Vienna, Austria.

Retired Fellow.

*1885. Charles H. Knight, New York City.

Active Fellows.

*1880. Harrison Allen, Philadelphia, Pa.
*1878. Morris J. Asch, New York.
1895. J. E. Boylan, Cincinnati, Ohio.
*1883. C. W. Chamberlain, Hartford, Conn.
*1882. S. Hartwell Chapman, New Haven, Conn.
1884. S. Solis-Cohen, Philadelphia, Pa.

1880. E. W. Cushing, Boston, Mass.
1878. Ephraim Cutter, West Falmouth, Mass.
*1880. W. H. Daly, Pittsburgh, Pa.
*1878. F. H. Davis, Chicago, Ill.
*1901. E. T. Dickerman, Chicago, Ill.
*1878. Frank Donaldson, Baltimore, Md.
*1878. William Carr Glasgow, St. Louis, Mo.
†1879. Edgar Holden, Newark, N. J.
*1882. Franklin H. Hooper, Boston, Mass.
1882. Frank L. Ives, New York City.
*1878. Hosmer A. Johnson, Chicago, Ill.
*1879. Woolsey Johnson, New York City.
*1880. William C. Jarvis, New York City.
1879. R. H. Kealhofer, St. Louis, Mo.
*1901. Gordon King, New Orleans, La.
*1885. Chas. H. Knight, New York City.
*1878. Rufus P. Lincoln, New York City.
1881. G. W. Major, Montreal, Canada.
1879. Charles McBurney, New York City.
1885. H. Clinton McSherry, Baltimore, Md.
*1881. E. C. Morgan, Washington, D. C.
*1886. J. C. Mulhall, St. Louis, Mo.
1892. T. Morris Murray, Washington, D. C.
*1881. H. Mynter, Buffalo, N. Y.
*1893. James E. Newcomb, New York City.
*1895. J. E. H. Nichols, New York City.
1879. H. K. C. Oliver, Boston, Mass.
1894. William H. Park, New York City.
1892. W. Peyre Porcher, Charleston, S. C.
*1880. D. N. Rankin, Allegheny, Pa.
1881. J. M. Robertson, Detroit, Mich.
1878. Beverly Robinson, New York City.
*1878. T. F. Rumbold, St. Louis, Mo.
†1880. Charles E. Sajous, Paris, France.
*1879. Carl Seiler, Scranton, Pa.
*1893. Charles M. Shields, Richmond, Va.
1879. Andrew H. Smith, Geneva, N. Y.
1879. Bernhard Tauber, Cincinnati, Ohio.
*1899. Max Thorner, Cincinnati, Ohio.

1888. S. O. Van der Poel, New York City.
1878. Clinton Wagner, New York City.
1896. Marshall R. Ward, Pittsburgh, Pa.
1879. Whitfield Ward, New York City.
*1886. Benjamin F. Westbrook, Brooklyn, N. Y.
1896. Green V. Woollen, Indianapolis, Ind.
1878. George M. Lefferts, New York City.
1901. Frank Hyatt, Washington, D. C.
*1885. Charles H. Knight, New York City.
*1880. C. E. Bean, St Paull, Minn.
*1893. Arthur Ames Bliss, Philadelphia, Pa.
*1878. E. L. Shurly, Detroit, Mich.

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*Deceased. †Corresponding Fellow. ‡Honorary Fellow.

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American Laryngological
Association
Transactions of the
annual meeting

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